PROGNOSIS IN GENERALISED PERITONITIS

--APACHE-II SCORE

Dissertation submitted to

The DR. MGR MEDICAL UNIVERSITY, Tamilnadu

in partial fulfillment of the regulations

for the award

degree in GENERAL SURGERY

Tirunelveli Medical College, Tirunelveli

The Tamilnadu Dr MGR Medical University,

Chennai, Tamilnadu.

APRIL 2013

DEPARTMENT OF GENERAL SURGERY

TIRUNELVELI MEDICAL COLLEGE

TIRUNELVELI

CERTIFICATE

Certified that consolidated dissertation "PROGNOSIS IN GENERALISED PERITONITIS – APACHE-II SCORE " presented here by Dr. PRAVEENKUMAR, is based on bonafide cases investigated and studied by the candidate himself in the wards of Tirunelveli medical college, Tirunelveli.

GUIDE

Prof Dr S Soundararajan,

Professor and Head,

Department of General Surgery,

DATE: TVMCH Tirunelveli

ACKNOWLEDGEMENT

It gives me immense pleasure to express my deep sense of gratitude and heartfelt thanks to my guide Dr. S Soundararajan, Chief and HOD of Department of General Surgery, Tirunelveli Medical College and Hospital, Tirunelveli for his kind help, expert advice and constant encouragement in preparing this dissertation.

My respectful regards and humble thanks to Dr. Manoharan, Honorable Dean of TVMCH, Tirunelveli whose ideology and principles have instilled a sense of discipline and responsibility in me.His kind advice as very helpful in conducting this study.

I would like to thank sincerely to Chief of other units Dr. Sreethar MS, Dr.Maheshwari MS, Dr.Pandy MS, Dr.varadharajan MS, Dr.Alex Edward MS, and Dr.Raju, Dr. Raj Kumar, Assistant Professors of Surgery, TVMCH, Tirunelveli for their constant support and valuable advice from time to time.

I am thankful to other staff of the Department of Surgery for their kind help.

No study can be completed without patients. I have been fortunate enough to have patients who were very co operative and i am highly indebted to them.

Last but not the least, i immensely thank my parents for their invaluable support and encouragement throughout this study

Place: Tirunelveli PRAVEENKUMAR

PROGNOSIS IN GENERALISED PERITONITIS

--APACHE-II SCORE

Dissertation submitted to

The DR. MGR MEDICAL UNIVERSITY, Tamilnadu

in partial fulfillment of the regulations

for the award

degree in GENERAL SURGERY

Tirunelveli Medical College, Tirunelveli

CONTENTS

INTRODUCTION

INTRODUCTION:

Peritonitis is the major cause for mortality and morbidity. Even though with adequate antibiotics coverage, adequate medical support peritonitis is supposed to be dominating cause for deaths.

Now-a-days since there is life style modification, sedentary work habits, higher calory intake, and consumption of alcohol and smoking have greatly increased chances of risk for mortality and morbidity.

Various disease can be evaluated with various clinical scaling for example Ransons criteria for acute pancreatitis. Similarly certain indices can be used for evaluation of generalised peritonitis. One such a method is APACHE-II scoring. This helps in assessing the outcome of patients treated with peritonitis.

AIMS

An study conducted on patients with generalised peritonitis with following aims: –

a) To evaluate the incidence of mortality rate in generalised peritonitis in TVMCH, Tirunelveli.
b) To implement APACHE-II in assessing the degree of severity of generalised peritonitis.
c) To find out the contributing factors for deaths in generalised peritonitis.

LITERATURE

PATHOLOGY OF GENERALISED PERITONITIS

DEFINITION:

Peritonitis is defined as inflammation of peritoneum due to contamination by the contents of gastrointestinal tract or the purulent material. It is essentially an interaction between the host and pathogenic micro organisms. Most of the cases, there will be microorganisms harbouring at the site of infection. It may be aerobic or anaerobic organisms.

CLASSIFICATION

Earliest classification was proposed by Meakins. It was based on anatomic areas of infection. There are 10 groups in the study.

GROUPS

I. From gastroesophageal junction up to ligament of treitz
II. Small intestine
III. Large bowels proximal to peritoneal reflection on rectum
IV. Post operative
V. Biliary tract
VI. Pancreas
VII. Appendix
VIII. Liver

IX. Gynaecological sites

X. Sites distal to peritoneal reflection on the rectum

PATHOGENESIS OF PERITONITIS

Space between the visceral and parietal peritoneum, the so called peritoneal cavity is a potential space prone for infection. The mesothelial cells which has the same embryonic origin as that of endothelial cells lines two surfaces .The surfaces consists of 3 layers from below upwards. A supportive layer of connective tissue, basement membrane and the flat mesothelial layer. The mesothelial cells gain a unique anatomical and functional arrangements where they overlay the diaphragm .

VON RECKLINGHAUSEN in 1983 described the so called stomatas. stomatas are the the gaps present in between the cells of the mesothelial layers and they are many in numbers and are found only in the muscular portion of the diaphragm. The functions of stomata are:

1) To allow water and electrolyte exchange
2) To absorb particles and transfer
3) To serve as entrance for lacunae of lymphatic channels present parallel to the muscle fibre of the diaphragm. The lacunar consist of valves which

prevent the backflow through them. There channels ultimately drain into thoracic duct via the substernal lymph node.

The peritoneal surfaces other than the diaphragmatic area are semi permiable in nature. Hence there is two way exchange of water and electrolytes through them.

MODE OF ACTION: on inspiration diaphragm contracts and descends down, this will cause increased intra abdominal pressures thus constriction of stomata. On expiration the stomata opens due to negative pressure and there is inflow of fluid from the abdominal cavity. The diaphragmatic muscle contraction propels the fluid through the duct.

Fowler discovered that the diaphragmatic lymphatic system acts as a great source of host defence to act against bacteria and hence he proposed that mortality due to peritonitis can be decreased by decreasing the bacterial absorption from peritoneum. This can be achieved by keeping the patient in upright position.

ROLE OF ADJUVANT FACTORS:

There are several chemical insults to the peritoneal surfaces. They include bile, gastric acid, pancreatic enzymes. Chemical peritonitis ultimately leads to bacterial peritonitis which is a common complication. Initially following chemical peritonitis there is a minor bacterial action over the area which creates a favourable environment for the bacterial multiplication.

Intraperitoneal fluid also acts as an adjuvant factor by inhibiting two important host defences:

a) By diluting proteins like immunoglobulins and complements
b) By making phagocytes incapable of digesting the bacteria by diluting them in the fluid.

In addition fibrin also acts as adjuvant by trapping bacteria and hence makes it impossible for the phagocytes to reach the bacteria. They also leads to premature degranulation while attempting to engulf the fibrin which leads to abscess.

Bile also acts as an adjuvant factor in potentiating bacterial infection, bile salts being the main compound responsible. Present experimental studies have shown that chemical toxins present in caecal contents are responsible for making them an adjuvant factor.

CELLULAR DEFENCES:

The normal cell count of peritoneal fluid is 3000cell/ml and they include mononuclear cells such as macrophages and lymphocytes .Neutrophils are typically absent.

Main function of macrophages is phagocytosis but they also secrete procoagulant factors which induces clotting via intrinsic pathway and helps in local bacterial entrapment, prostaglandins and leukotrines

(neutrophill chemoattractant) other then macrophages,there are also eosinophills, basophills , and mast cells are presentin peritoneal cavity normally. Large amount of histamine is present in basophills and mast cell granules which being an inflammatory mediator causes vascular dilatation and endothelial cell contraction by increasing vascular permeability. This causes influx of large amount of fluid and plasma proteins that includes complement and immunoglobulins. In case of peritonitis, localisation of infection occurs due to inhibition of fibrinolytics activity and formation of fibrin adhesions will lead to localisation of infection.

PERITONEAL REACTION TO INFECTION

Peritoneum initially reacts by releasing numerous non specific inflammatory mediators. Macrophages and mast cells release prostaglandins and histamine which causes vasodilatation. This leads to increased vascular permeability and exudation of immunoglobulins, fibrins, clotting factors and other factors. Initial 4-6hrs there will be influx of neutrophils due to liberation of chemotoxins like leukotrine B4 and C5a. Further this causes a series of inflammatory pathway in phagocytes and complement system tries to prevent infection.

There are 3 important mechanisms which helps to remove bacteria from the peritoneal cavity in the early phase of infection

a) Diaphragmatic lymphatics

b) Macrophages in peritoneum

c) Neutrophilic influx

In responce to inflammatory stimulus, mesothelial cell retraction and stomata of diaphragm enlarges which increases patency of numerous stomata which may lasts as long as more than 3days. This increases uptake of bacterias.

In the next phase bacterias gushes into the systemic circulation leading to septicaemic phase, however neutrophils tries to resist bacterial proliferation in this phase.

What is important here is the neutrophil proliferation (influx) is not dependent either on bacterial size nor bacterias alive/dead. Hence this concludes that neutrophil proliferation is not sufficient to combat the infection and requires other host defence systems.

Portal venous systems also found to play key role in bacterial elimination. According to Glofsson et al, there was increased level of endotoxins in portal venous arcade within 10 minutes of peritonitis. This endotoxin acts on kupfer cells and causes hepatic malfunction.

BACTERIMIA IN PERITONITIS:

Gastrointestinal tract pathology is the main cause for peritonitis, others include abdominal trauma, post surgical nosocomial infection.

Often it was observed that level of perforation in GI tract will give clue for the type of organisms. Gastric perforations are associated with gram positive anaerobes, candida, rarely gram negative bacilli. Colon and terminal ileum are associated with most commanly facultative anaerobic organism E.coli however lower GI contains more than 400 different types of microorganisms and concentration of 10^{12}/gram. Other organisms found in the flora are klebsiella, proteus and pseudomonas, enterococcus, peptostreptococci, clostridia and bacteroides fragilis. Bacteroides fragilis is found to be increasing incidence in peritonitis.

Weinsten et al conducted a series of studies and did an experiment to prove colonic peritonitis by keeping a rats caecal material with barium into peritoneum

First 4-5 days:- acute gen.peritonitis which lead to mortality of about 40% and in this phase 95% of animals had E.Coli bacteremia.

Second phase: - multiple abscess in the peritoneum and no further deaths. However peritoneal culture revealed E.Coli, Enterococcus and bacteriology revealed 27 species in the culture from original inoculums. Thus it should be noted that E.Coli and Enterococcus

mainly dominated in the peritonitis stage and Bacteroides fragilis in the abscess stage.

Synergetic action of bacteria is also important. Intraabdominal infection has both aerobic and anaerobics. In an experiment in pure culture media lab animals can tolerate microbial organisms but when synergetic they will not tolerate. Anaerobes may play important role in forming abscess but their role in developing toxicity is questionable. For example Bacteroides fragilis alone is innocuous since its lipopolysaccharide is ineffective but it can potentiate the pathogenic property of other aerobes in different infection. Thus in peritonitis B.fragilis potentiates the toxic effect of non lethal E.Coli.

Studies conducted by Rotstein et al proves this point that B.fragilis and other anaerobes secretes heavy dose of succinate which inhibits and interferes in neutrophilic function.

SUMMARY:

Pathologicaly the coliform and B.fragilis play important role in peritonitis.

E.Coli appears to cause early deaths in first 4-5 days.

Anaerobes like B.fragilis is major cause for abscess formation.

MULTIORGAN DYSFUNCTION SYNDROME

Definition:- abnormal dysfunctioning of two or more systems on the body. This is the end stage for any inflammatory pathology in body and carries highest mortality. It was observed that MODS occurs in 5-20% in emergency surgeries and around 30% -50% of peritonitis.

With the advancement in medical technology and advanced intensive care the MODS has reduced to a certain extent. As the number of organs involved, chances of mortality also increases. Involvement of two major organs carries mortality of 60% and three or more organs about 80% and more than four organs 100% mortality.

FACTORS INFLUENCING MODS:-

Sepsis is the major cause however other causes include trauma, hypovolumic shock, extensive tissue necrosis due to burns or major surgical procedures. Septicemic patients are more prone for MODS.

In the recent years, the role of intestinal barriers has been established. The intestinal mucosa normaly prevents the escape of endotoxins and microorganisms from the intestinal lumen. Failure of intestinal barriers results in escape of microbes from the intestinal lumen. This causes bacterial

translocation. Hence it can be easily explained that in case of hypovolumic shock, burns/infections MODS is caused by this bacterial translocation.

Eiseman et al studied 42 patients with MODS. Sepsis was the main factor in 69% of cases that died.

Fry et al study on 38 patients of MODS showed 74% of mortality rate. This indicates that

1) Incidence of MODS – 7% in post surgical cases.
2) MODS occurred mainly due to infection.
3) MODS occurred in sequences – lung, liver, gastric mucosa, kidney.
4) MODS is the final outcome for any uncontrollable infection.

SYSTEMS INVOLVED IN MODS:-

Cardiovascular system, respiratory system and renal systems are the major organs involved. Curling ulcers with bleeding may be considered the cause for GI tract dysfunction. In blood DIC is main reason and lastly CNS system is affected.

In common practice lab values and signs and symptoms may be used to describe organ dysfunction. Here are the criterias which can be used to asses organ dysfunction.

1) Respiratory system:- Respiratory dysfunction may be defined when patient requires ventilator support more than 5 days and Fio2 more than 0.4.

   This criteria will detect truly hypoxic patients who are in need for respiratory support

2) Hepatic system:- Hepatic dysfunction is defined as hyperbilirubinemia more than

2.0mg/dl and elevated SGOT and LDH greater than three times the normal.

The enzyme levels are also included in the criteria in order to exclude the transient hyperbilirubinemia resulting from retroperitoneal hematoma, pelvic fracture or jaundice due to blood transufusion.

4) Renal system:- renal dysfunction is defined as increase of serum creatinine more than 2mg/dl. Urine output is excluded from the criteria since renal dysfunction may also some times associated with polyuria as occurs in multiple traumatic injuries.
5) GIT systems :- GIT dysfunction is defined with respect to upper GIT bleeding and its association with curlings ulcer i,e stress ulcers of stomach in major burns. Endoscopic documentation of GI bleeding is excluded. Thus upper GI bleeding whose blood transfusion requiring

more than 2 units is included for definition. Thus Fry et al used above said criteria for patients undergoing emergency surgical procedures in 553 pts and reviewed. Respiratory , hepatic, renal dysfunction occurred almost at same frequency around the range of 8%

Bleeding due to stress ulcer occurred in 3%

2 or more organ failure occurred in 38 patients with mortality rate 74%.

MECHANISM OF MODS:-

Certain pathogenesis is responsible for MODS .septicaemia is the major stimulatory factor which destabilises haemodynamics and metabolism. In sepsis cardiac output increases 3 times the normal, the peripheral vascular resistance decreases, peripheral utilisation of O2 decreases and A-V O2 concentration difference is narrowed .These changes ultimately lead to lactic acidosis leading to hypovolumia and hypotension.

Further more there will be increased hepatic glucogenisis and increased nitrogen excreation in urine. Hepatic glucogenesis requires carbon atom which is released from muscle breakdown and by deamination urea is excreted.

Above explanation gives clue for the organ dysfunction and septicaemia ;

1) Origin of organ dysfunction starts from periphery and not due to impaired cardiac function.
2) A common tissue trauma leads to altered physiological state.

Thus above said clues helped scientist to keep their school of thought of involvement of humoral factors which leads to metabolic dysfunction and ultimately organ malfunction.

Prostaglandins:- there are more than ten components described. Each component has its own role. Important ones are.

Prostacyclins:- it is a vasodilator and will prevent platelet aggregation.

Thromboxane:- it is vasoconstrictor and enhance platelet aggregation.

These two componant play key role in sepsis and MODS.

Interleukins:- macrophages secretes IL-1 by stimulation by bacteria.

Function:- lympocytic proliferation ; muscle proteolysis, hypermetabolism and enhances catabolism of MODS.

Fry et al studies shows that excess thromboxane may damage liver and lead to organ dysfunction thus mortality.

CATABOLIC HARMONES:-

These are stress hormones which are released from body in response to major trauma, infection, major surgery. These hormones include

hydrocortisone, glucagon, epinephrine. Recent study shows that GH and Thyroid hormone also acts as stress hormones. These stress hormones mediates MODS.

COMPLEMENT ACTIVATION:-

They are the plasma proteins activated in response to foreign antigen. Alternate pathway activated by bacterial endotoxins play important role in MODS.

After complement activation by antigen there will be release of C3a and C5a which promotes margination of neutrophils by chemoattraction to the site of contamination. Further there will be release of anaphyllotoxins and histamine which causes increased vascular permeability. However this complement activation might be beneficial to local site but for systemic infection it is hazardeous.

Many experimental studies have proven that complement activation hazardeous to human physiological systems.

OXYGEN RADICALS:-

Biological membranes are more vulnarable to lethal effects of charged O2 radicals. These are produced by phagocytes for destruction of bacteria. Abnormal production of O2 radicals occurs by two mechanisms.

1) Inappropriate margination of activated neutrophills.
2) Cellular hypoxia

They are said to damage tissue and finally MODS.

OPIOIDS:-

These are released in Central nervous system during stress. It includes enkephalin, endorphin, dinorphin, and endogenous opioids. They causes sepsis and MODS.

FIBRONECTIN:-

They are nonspecific opsonin.

Function:- they bind themselves to foreign particles and are cleared by kupfer cells.

Fibronectin malfunction may cause microembolisation leading to tissue ischemia and MODS.

OTHERS:- such as TNF and kinin also play role in septicaemia and MODS.

THOERIES: MICROCIRCULATION AND PERFUSION FAILURE

As discussesed initially, it comprises increased cardiac output, decreased peripheral vascular resistance, narrow A-V o2 difference which leads to lactic acidosis and hyovolumia and hypotension.

What it implies is that defective in utilisation of o2 at the peripheral region is main cause for organ failure. If we consider at the intracellular level there is abnormal oxidative phosphirylation and decreased O2 metabolism.This major events will be taking place in mitochondria.

Surprisingly, Fry et al did not mention about mitochondrial abnormality in his experiments rather he stated that septicaemia occurs due to defective microcirculation in visceral organ. This defectiveness leads to abnormal cellular energy. However skeletal muscles, which does not take part in microcirculatory defects are hyperperfused and viscera are hypoperfused in hyperdynamic situation.

Further different experiments showed the importance of visceral perfusion.

Trauma, endotoxins and foreign antigen causes complement activation and production of C3a and C5a which inturn leads to

platelet aggregation, margination and increased production of prostaglandins.

Abnormal proliferation of neutrophils produce O2 free radicals. These radicals damage the endothelium, platelet aggregesion, and release of thromboxane.

All these events are serialy noted in ARDS, liver, gatric mucosa. Hence indirectly showing the importance of microcirculation and biochemical blockage.

FACTORS INFLUANCING THE PROGNOSIS OF GEN.PERITONITIS

Various factors are implicated. They are;

1) Age
2) Chronic disorders such as Diabetes mellitus, respiratory disorders, renal disorders
3) Time duration
4) Infective source
5) MODS

AGE:-

As the age increases immunity decreases and host interaction decreases due to decrease in the number of phagocytes to fight at the site of infection.

Below are the points noticed as the age of humans increases :

a) Reduction in size and mass of thymus gland. It causes reduced number of mature T-cell.
b) Reduced chemotaxis and phagocytic action of leukocytes.

Studies conducted by Bohenen and Boulanger showed that in generalised peritonitis age less than 50yrs have 17% mortality and more than 50yrs of age have 45% mortality.

Post surgical patients developing peritonitis have no correlation between age and mortality. Hence age is not said to be important criteria for post surgical peritonitis.

Pine et al studies showed that death in his cases were 27%. Mean age for survivors found to be 47.5 ± 21yrs (range 47 – 94yrs) and patients died were 66.5 ± 10.5 yrs (range 47-81yrs). It was also noticed that proportionality between age of humans and organ dysfunction.

Dawson et al noticed that age less than 50 yrs in diffuse peritonitis carried mortality less than 10%. As the age increases death rate increased and in more than 70 yrs patients died of secondary peritonitis with a mortality rate almost 100% except appendicitis.

According to Kalfarentozos et al, age more than 65yrs mortality rate was 33% in severe peritonitis.

According to Fry et al (studies over 143 patients with peritonitis) age group more than 50yrs in 50 patients, 22 patients had died.

Bohnen et al conducted a prospective study in 100 patients suffering with generalised peritonitis and found that:

Age group more than 65 yrs – mortality was more than 38%

Age group less than 65yrs – mortality less than 27%

Survivors mean age was 57.8yrs and those who died had mean age of 60.8 yrs.

Studies by Hunt in 54 pts in generalised peritonitis :-

Dead patients- average age 62 yrs

Survived patients- average age 49yrs.

Studies by Dellinger et al over 187 patients showed age of 64+_14 carried mortality of 24%.

Hence different studies have conclusively proved that old age carries higher rate of mortality in peritonitis.

DIABETES MILLETUS:-

It is pancreatic exocrine disorder. It has potential effect on host defence. It disturbs the host defence against the foreign

antigens and decreases the phagocytic property of leucocytes.

The level of blood glucose affects the phogocytic property of leukocytes both at higher and lower level.

Studies done by Kalfarentezos et al in 42 cases of peritonitis showed that DM is an important element. One out of 16 patients with DM died with peritonitis and carried mortality of 66%

Studies done in 187 patients by Dellinger et al showed that DM is an important risk factor with $p < 0.005$. In their study they found that 10 patients out of 16 patients having DM died.

Pine et al in his study noted that DM is not an important factor. In their study 5 patients of peritonitis had DM and no one died.

It still remains questionable that whether DM is an important risk factor for patients with peritonitis.

3) TIME DURATION :-

Pre operative evaluation of duration of disease is an important aspect in terms of a disease mortality and outcome. As the time duration increases patients are set to go for septic shock and growth of harbouring gram negative bacteria and others microbes.

Hunt did study on 44 patients and reported that mortality rate is two times greater in patients who underwent surgery for peritonitis after 24 hrs of onset of symptoms of peritonitis than in with less than 24hrs.

Less than 24hrs- operated- mortality 17.6%

More than 24hrs- operated – mortality 50%

Bohnen et al study shows earliest surgery less than 24 hrs have good prognosis, and more than 24 hrs carry higher mortality upto 61%.

4) INFECTIVE SOURCE :-

Infective source is essential factor in determining the prognosis in peritonitis. Mortality rate in duodenal perforation and gastric perforation is 9-40%.

Dawsons study showed 20% mortality in duodenal perforation and 46% mortality in gastric perforation.

Onodera et al noted 25% mortality in peptic ulcer perforation in his study.

On the other hand mortality rate in peritonitis caused by small bowel pathology including obstruction and strangulation was found to be 20-25%

Large bowel pathology carries higher mortality. Studies conducted by Miller and Wichern in 118 cases found that colonic perforation in colonic carcinoma carries 30% mortality.

Bohnen et al study in 176 patients, out of which 67 patients died with mortality of 38%. The study was conducted by dividing patients into 3 groups.

Group I :- includes 68 patients , they were diagnosed to have DU perforation and appendicular perforation. Out of which 40 pt having DU perforation and 28pt having appendicular perforation. This group showed mean age of 49yrs with mortality 10%. 7deaths were due to DU perforation. No deaths observed on appendicular perforation.

Group II:- had 48% patients , the cases selected in such a way that perforation of intraabdominal organs except duodenum and appendix. Mortality was found to be 50% and mean age 63yrs.

Group III:- 60 patients selected, selected group were post operative peritonitis and it carried mortality of 60%

Stephan and loewenthal study report says that out of 68 patients having gen.peritonitis, 33 pt died. The death was mainly due to old age, post surgical anastamotic leakage, wound dehinscene an faecal peritonitis.

Hunt et al studies showed that mortality rate varied according to site of infectious origin.

Gastric/ Duodenal perforation – 12.5%

Small bowel-21%

Colon- 54%

Others- 40%

Meakins et al thus proposed an classification for intraabdominal sepsis based on anatomical and physiological unit .These are 10 types which has been already discussed .

5) MODS :-

MODS carries higher mortality rate and almost 100% death rate has been observed.

It is charecterised by multiorgan system failure.

It was shown in 7-22% of emergency surgery procedure and 31-55% cases with bacterimia or abscess inside the peritoneal cavity.

Retrospective study done by Fry et al on 533 patients who underwent emergency surgery, the mortality rate in was found to be as follows :-

No organ failure – 3%

One organ failure – 30%

Four organ failure- 100%

Machiedo et al study:- He reported the aetiology for death in surgical intensive cares and showed that 20% patients had septic foci in the abdomen and 30% had organ failure.

Bohnan et al study showed that patients with MODS had higher death rate of more than 76%. The operative procedures were delayed in the organ dysfunction. Early surgical operations in an organ failure patients showed better prognosis compared to delayed surgical operation.

Hence patients who had not undergone surgery within 24 hrs of organ failure had 88% mortality.

Sweet et al observed that 14-17% death rate occurs in intensive care unit due to lung insufficiency or kidney dysfunction. Mortality rate was found to be 65%. The same mortality rate noticed with massive peritonitis and acute kidney dysfunction and mortality rate of 80% found with lung insufficiency and generalised peritonitis..

SCORING SYSTEM FOR ASSESING GEN.PERITONITIS

Stevens et al in 1983 made an important devise which gives quantitative severity of peritonitis. The main principle is progression of organ dysfunction in one or many organs due to septicaemia. In order to achieve the scoring in patients having sepsis, various organs have been given numerical values depending on available clinical and physiological datas. He defined vital 7 organ system and assigned a score ranging from 0 to 5 in each system.

The calculation was done in the form of SEPTIC SEVERITY SCORE in which squaring of values for every organ system and three highest scores were added to achieve final score. He proposed that squaring of individual scores exponentially increases mortality

with progressive multiple organ dysfunction. The range of score was 0-75. In his paper with sample study of 35 patients, the patients who scored 40 had 82% mortality (9 patients of 11) as compared to 2% mortality (4 patients of 19) for patients scoring below 40.

Further studies were done by transfusing fibronectin in generalised peritonitis. Steven et al studied on 31 samples with scores from 9 to 71. The 89% mortality rate was found in those having score more than 40 and 32% mortality with samples having less than 40. Suppose 40 was cut off value to asses the death, the sensitivity comes to about 77% for both study that is 47 of 61.

Skan et al studied on 58 patients having generalised peritonitis and they made comparision between septic severity score of stevens and acute physiology score. Thus skan et al modified the definition in septic severity score so that subjective phenomena can be reduced. Further they showed a correlation coefficient by increasing score and increased mortality.

Elebute and stoner in 1983 gave device to grade the severity in septicaemia. According to this septic patients were classified into 4 classes depends on clinical features and degree of severity upon analogues scale. These includes degree of temperature elevated,

local effect of tissue infection, secondary effects of sepsis and laboratory values.

The scoring ranges between 0-45. This depends on interpretation of tables.

Dominion et al further undertook study on this system. They studied on 135 patients with a number of various infection such as pneumonia, peritonitis, urinary tract infection, wound site infection, septicaemic patients, abscess. The score for septicaemia was 10-30. They saw overall mortality rate of 56% and a score below 20 had 20% death (13 dead from 64 patients) and score above 20 had 89% death (63 dead from 71 patients). The score 20 was roughly chosen for assessing the death. Thus the sensitivity and accuracy by this method was 84% that is 114 patients of 135.

Since from the time it is well established that numerous serum proteins are released during acute infection. These are called acute phase proteins. They are stress hormones released in response to tissue trauma.

Dominion et al studied in 135 patients and saw the levels of acute phase proteins, alpha-1-antitrypsin, alpha-1-glycoprotein, and complement factor B. They studied these plasma level in who scoring work was done according to septic score of Elebute and

Stoner. To start, the concentration of acute phase protein studied in patient diagnosed as septicaemia and monitored serially during the course of illness. The results found that patients who survived had increased level of A1AGP, A1AT, FB and C3 where as non survivors had low levels.

Kalfarentzos studied in 42 patients. They monitored the physiology of liver, kidney, and coagulation factors, respiratory system, cardiac system, and CNS system with the help of stevens septic severity score. The reports found that gradings were variable. Grade III dysfunction had higher mortality rate especially respiratory dysfunction, cardiac dysfunction, coagulation abnormality.

ACUTE PHYSIOLOGY SCORE:-

Knaus et al in 1982 devised a scoring system to classify patients who were admitted in intensive care unit. They found that there was a need for additional parameters while concluding the accuracy of disease severity. They promoted two parts scale. This includes:-

a) Acute physiologic scoring -34
b) Chronic health evaluation.

a) ACUTE PHYSIOLOGIC SCORING can determine the pathology in patients at the 1st day of admission in the intensive care units. Numerical datas ranging from0 to 4 were given and higher the physical measures, the higher the abnormality for each patients. The overall score will be high. It ranges from 0 to 124 and this scoring signifies the capability of patients haemostatic function to tolerate the acute illness.

b) CHRONIC HEALTH EVALUATION:- in this case we take detailed health status of patient before admission and any possible comorbidity, functional capability, and any medical check ups in the past 6 months before admission. Thus on the basis of history and question answer session patients are categarised into Catogary.A,B,C,D.

Thus two part scales APS 34 and CHE together called APACHE. It should be noted that APACHE was not basically designed for severe surgical site infection during initial days of its introduction.

A very selected group of patients were selected during its initial study. As during the course of time APACHE was rapidly used in many countries including Europe, USA. It

was found that APACHE is most reliable tool in evaluating risk for mortality in intensive care units.

Meakin et al in 1984 reported the study with the help of combining APS-34 and anatomic classification of origin of intraabdominal sepsis. They considered 187 patients for this system. It was an astonishing point noted by Meakin that there was a strong relation between increased mortality rate with increase in acute physiology scoring, during the time of infection being diagnosed. However in it there was additive risk factor present such as DM, shock. These additive factors never gave substantiating information to predict the survival rate and death rate once the physiologic scoring is considered. The report also found an correlation between age and malnutrition with mortality rate. It should be noted that the amount of local sepsis such as local peritonitis or abscess and anatomic origin of infection does not affected the prognosis provided the APS is under control.

Skau et al study showed comparision between APS and stevens septic severity scoring in 58 patients having peritonitis. It was noticed that there was good relation

between them and degree of correlation between them was 0.81. It was also found that age had potential effect on mortality rate. As the age increased, the mortality rate increased.

And further Andras et al did work on APS on the generalised peritonitis. Comparison was done between elective reoperation with mandatory reoperation. As such there was no difference noted. APS scale was used to asses the mortality rate in separate groups and for combined groups. The results were same as reported in previous studies.

APACHE –II SCORE:-

Classical APS had 34 laboratory values or physical tests and whenever there was unavailability of results it was presumed to be normal. Hence there was greater reduction in the requested datas. It is specially to be mentioned that APS was used only for intensive care unit patients and was practically not used for patients not admitted in intensive care unit.

In order to overcome these disadvantages Knan et al devised APACHE – II in which only 12 values are used for physiological measurements.

APACHE –II is more reliable for patients not admitted in intensive care unit. It is also more reliable scoring technique than APS scoring of Elubute and Stoner, Stevens.

APACHE – II is objective scoring and it is valid scoring in variety of cases and large amount of cases.

APACHE –II score has three parts devised by Knans et al :-

Part A:- APS having 12 laboratory investigations and physical findings (APS – 12 ; 0-60 points)

Part B:- Age group and points above 44yrs (0-6 points)

Parts C:- An chronic health; points are 0, 2 or 5 based on chronic health and whether the point is post surgical or not.

Bohnen et al in 1988 discovered APACHE –II system can be used for assessing the mortality rate in generalised peritonitis. The prospective study done in 100 patients with peritonitis. They evaluated APACHE-II for pre surgical stage of peritonitis. They found that mean score was 13.72 with range of 0 to 36. The mean score for those who died was 18.9 and mean score for those who survived was

11.4.when APACHE –II score increased, the mortality also increased.

It was also noted that 19 patients were receiving steroid on long term basis. Out of them 12 died. Patients receiving steroid at any point of time 16 died of 25 pt.

It was also noted a minor difference between post surgical peritonitis and spontaneous peritonitis with respect to mortality rate. This was a contrary to previous studies in which post surgical peritonitis carried 60% mortality rate. But these findings matched Dellingers et al study in which post surgical peritonitis was not associated with increased risk.

METHOD OF STUDY

AND

MATERIALS

The APACHE –II study was done in tirunelveli medical college and hospital, tirunelveli. The main aim was to study the risk factor, aetiology, and APACHE – II scoring.

In this prospective study we used 100 patients suffering with generalised peritonitis who were admitted in general surgical wards. The study was undertaken between 12/08/2011 to 12/08/2012.

Selection criteria done by random samples. The selected patients are all established cases of peritonitis which includes are gastric perforation , duodenal perforation, small bowel perforation, large bowel, appendicular perforation ,post surgical leakage, liver abscess, pancreatitis and others using variety of clinical data, ultrasound guided findings of intraabdominal collection and post surgical collection of free pus or gastric / intestinal contents in the abdomen. Thus we made conclusive diagnosis based on following datas.

Cases of appendicitis are added in this study provided there is presence of free fluid inside peritoneum with peritonitis. Genitourinary and gynaecological cases were excluded from the study.

These patients were treated intensively with antibiotics which covers aerobic and anerobic organism. From 100 cases we took 91 cases for emergency operation. We managed 9 cases conservatively by keeping bilateral flank drain because 3patients were not willing for surgery and 6 patients were unfit for

operation. Among 91 patients operated 4 patients died and out of 9 patients not operated 6 patients died and 3 patients recovered.

Aetiology for generalised peritonitis was studied in 10 groups by using Meakins et al classification.

For every patient, a record was made of findings on history and physical examination, laboratory data and all other test or examination s performed. All the diagnostic , clinical and laboratory characteristics were noted prior to the commencement of treatment especially before surgical intervention. If a particular variable was never measured, it was considered normal. The time and type of operative treatment were recorded, as were details of antibiotic therapy and were cultures of peritoneal fluid. The source of contamination was recorded as noticed on exploration.

Onset of the presenting illness was taken as the time when patient developed acute symptom like pain abdomen and duration of illness was taken as the time that elapsed from the time of onset to the commencement of treatment, especially surgical treatment. An APACHE score was computed for each patient on the day of admission before surgery.

The APACHE II score records the degree of deviation from the normal of 12, routinely measured laboratory tests or physical findings, using skill from 0 to 4. In normal test not obtained because it was not considered clinically relevant for the individual patient, was scored zero.

The risk factors included in this study were age, diabetes mellitus, duration of illness, source of infection or cause of intra abdominal sepsis and APACHE II score.

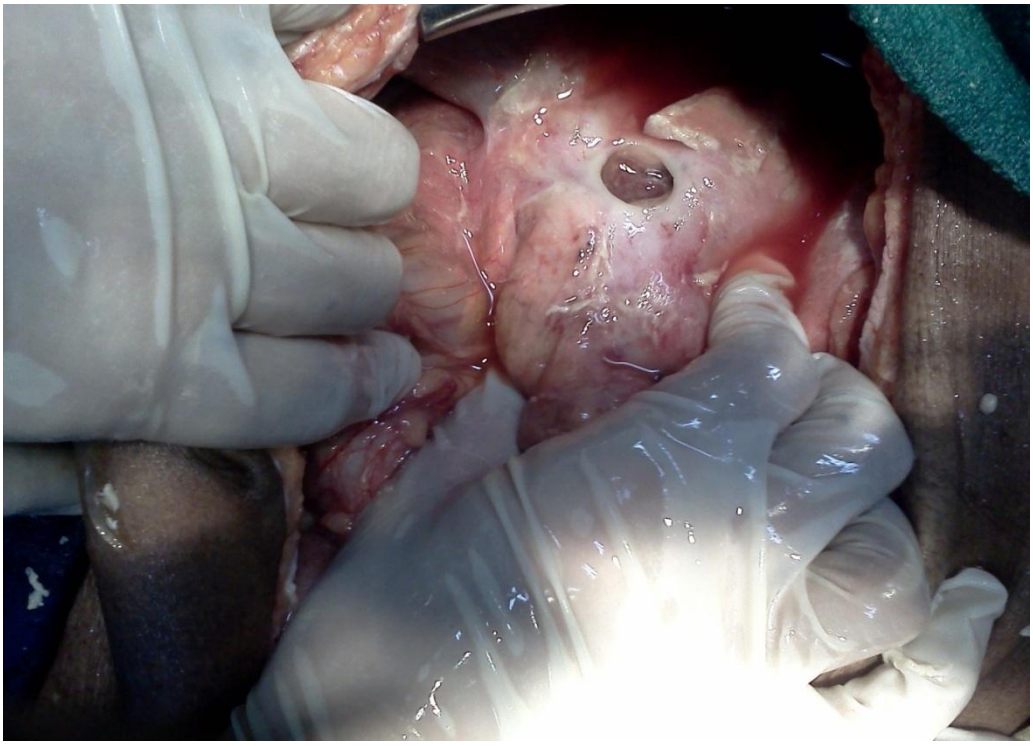


PHOTO:- A case of Duodenal Perforation

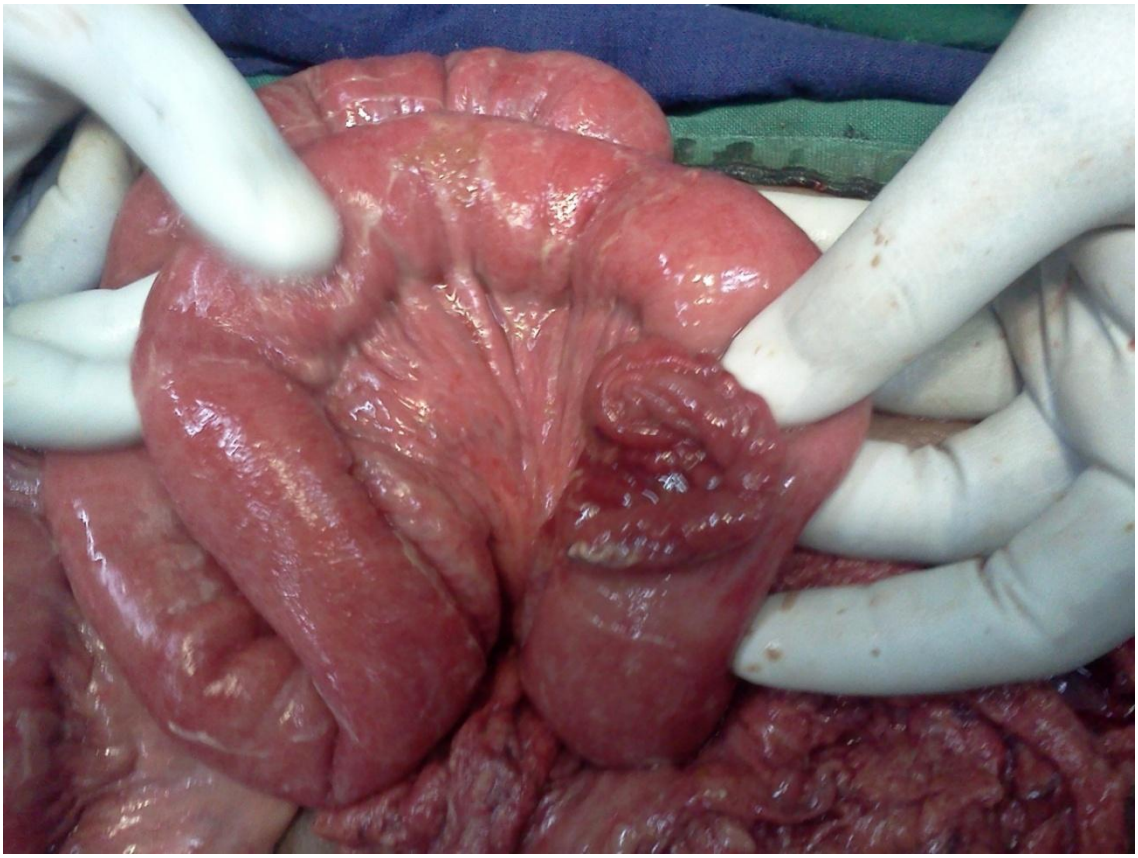


PHOTO:- A case of Ileal Perforation with Faecal fistula

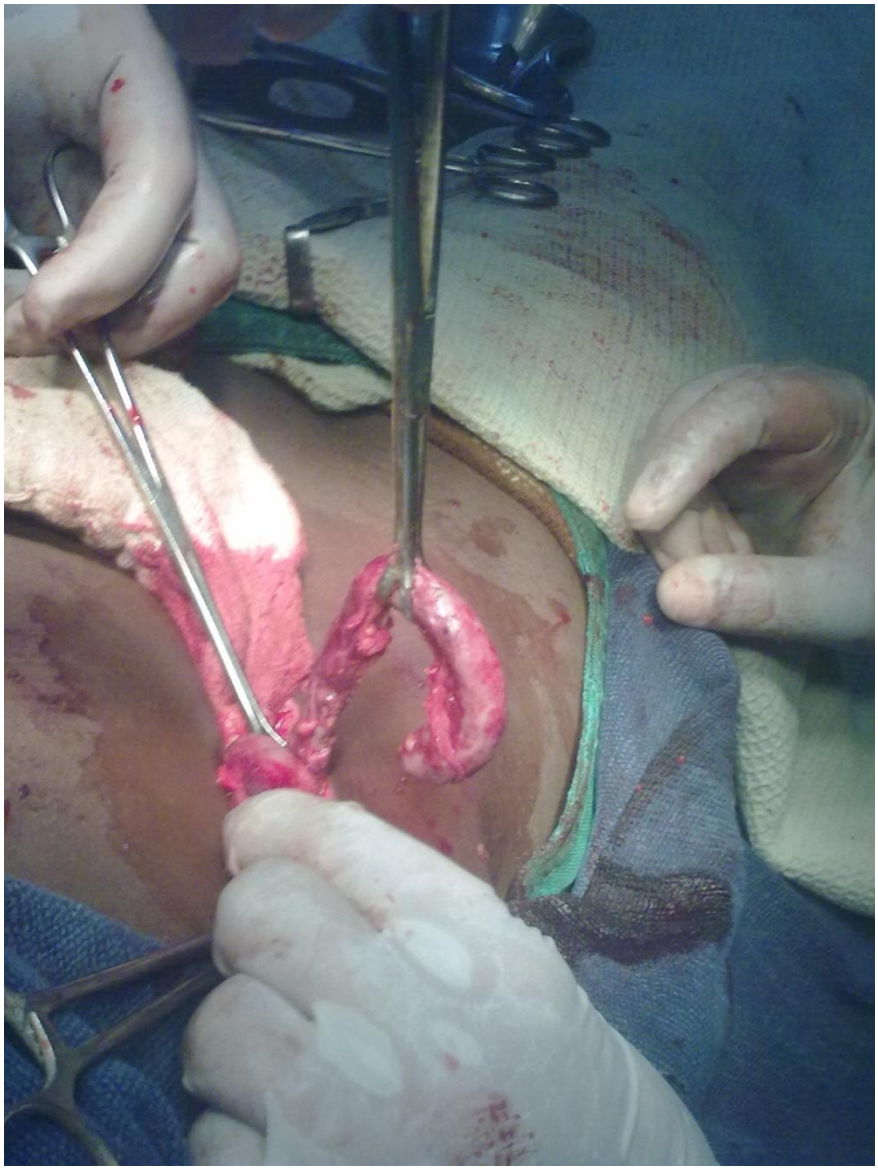


PHOTO :- A case of Appendicular Perforation

RESULTS

AND

ANALYSIS

This study includes 100 patients with intra abdominal sepsis (86 males and 14 females). The overall mortality in this study was 10%.

Table No. 1 shows the Age and Sex distribution of the cases in the present study.

Males accounted for 86% of the cases while females accounted for 14%, the sex ratio being 6.1:1 (M:F).

The maximum number of patients were In the age group of 30-39 years (31%) followed by those in 40-49 years group (20%) and 20-29 years group (17%)

Table I

Age and Sex distribution of the Cases

Age in years	Males	percentange	Females	Percentage	Total	Percentage
10-19	5	5.8	1	7.1	6	6
20-29	15	17.4	2	14.3	17	17
30-39	28	32.6	3	21.4	31	31
40-49	17	19.8	3	21.4	20	20
50-59	12	13.9	2	14.3	14	14
60-69	5	5.8	1	7.1	6	6
70 and above	4	4.7	2	14.3	6	6
Total	86	100	14	100	100	100

Table II shows the mean age and Standard Deviation of the cases according to sex.

Fig.I – Age and Sex Distribution of the Cases

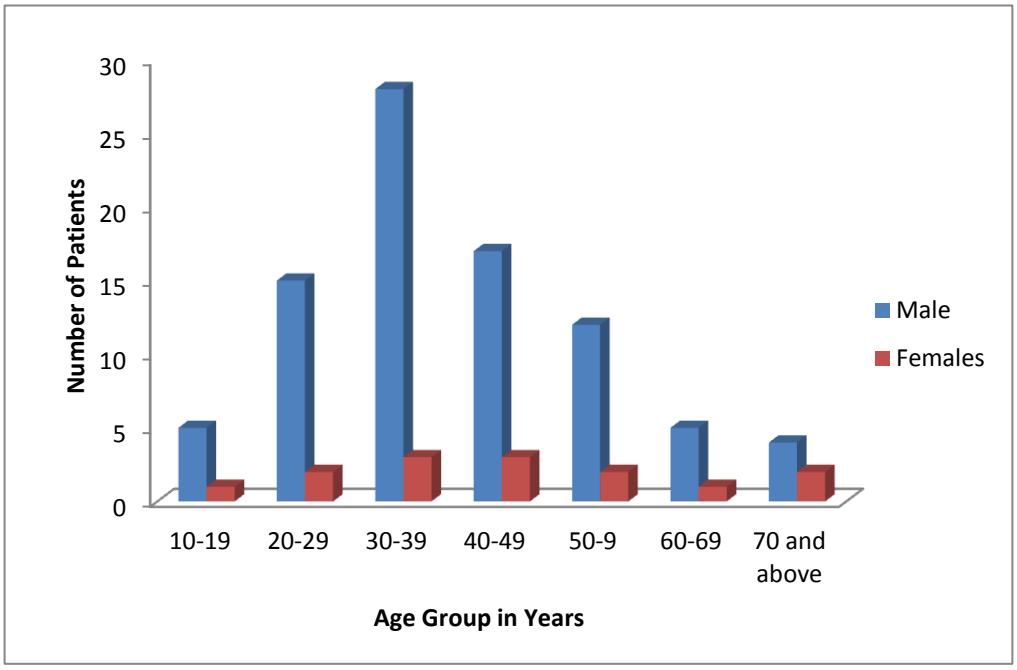


Table II

Mean Age and Standard Deviation Of the cases according to Sex

	Males	Females	Total (M + F)
Mean (yrs.)	39.9	44.5	40.6
Standard Deviation (yrs.)	14.6	17.7	15.2

From the above table it can be observed that the mean age among males was 39.9 years with a Standard Deviation of 14.6 years, while among females the mean age was 44.5 years with a Standard Deviation of 17.7 years. The difference in the mean age was statistically significant ($P < 0.05$) for both sexes put together.

The overall mean age of the patients in this study was 40.6 years while the Standard Deviation was 15.2 years.

AGE OF THE PATIENT AND CASE FATALITY RATE

Table III shows case fatality rate according to age and sex of the patients.

TABLE – III

Table showing CASE FATALITY RATE according to Age and Sex

Age in years	Males	Deaths	CFR %	Females	Deaths	CFR %	Total Cases	Total Deaths	Total CFR %
10-19	5	1	20	1	0	0	6	1	16.7
20-29	15	0	0	2	0	0	17	0	0
30-39	28	1	3.6	3	0	0	31	1	3.2
40-49	17	4	23.5	3	0	0	20	4	20
50-59	12	0	0	2	0	0	14	0	0
60-69	5	2	40	1	0	0	6	2	33.3
>70	4	1	25	2	1	50	6	2	33.3

The mean age for those who survived was 38.6 years compared to 49.7 years for those who expired.

The CFR among males was maximum in the age group of 60-69 years constituting 40% while among females it was 50% in the age group 70+ years.

The maximum total CFR was observed in the age groups of 60-69 years and 70 or more accounting for 33.3% each followed by patients in age group of 40-49 years accounting for 20%. Patients in age group of 10-19 years had a CFR of 16.7%.

Fig II – CASE FATALITY RATE in males according to age and sex

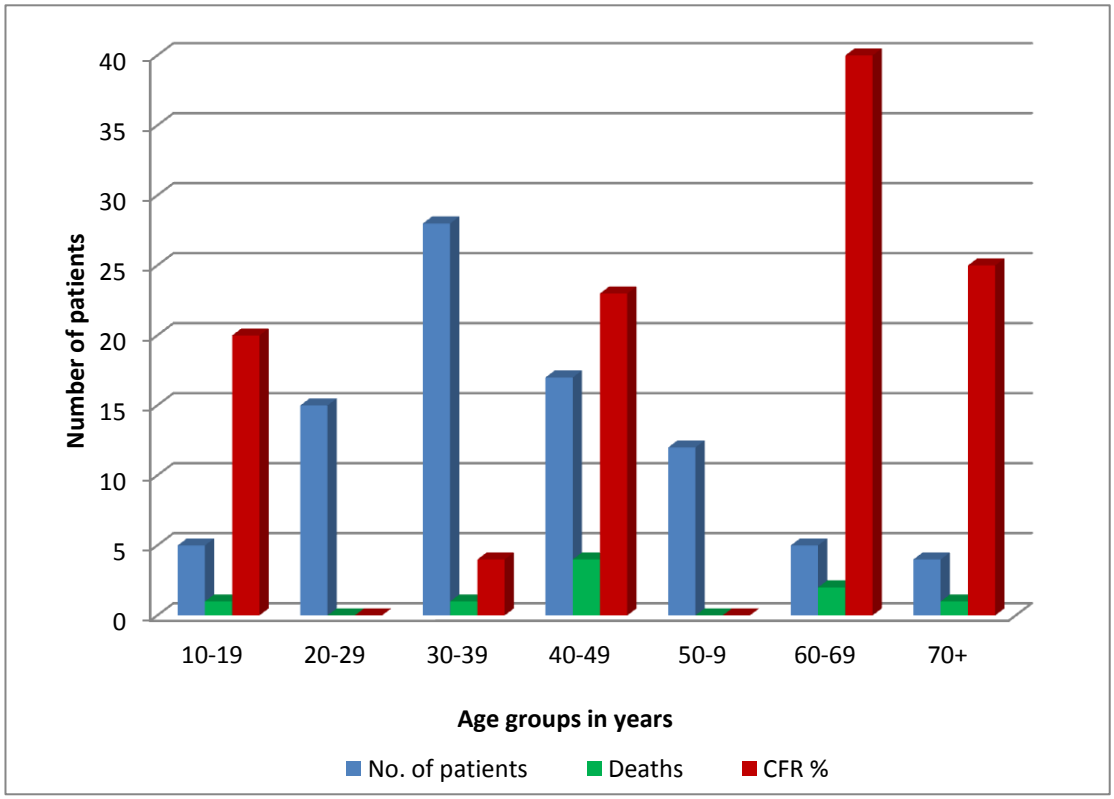
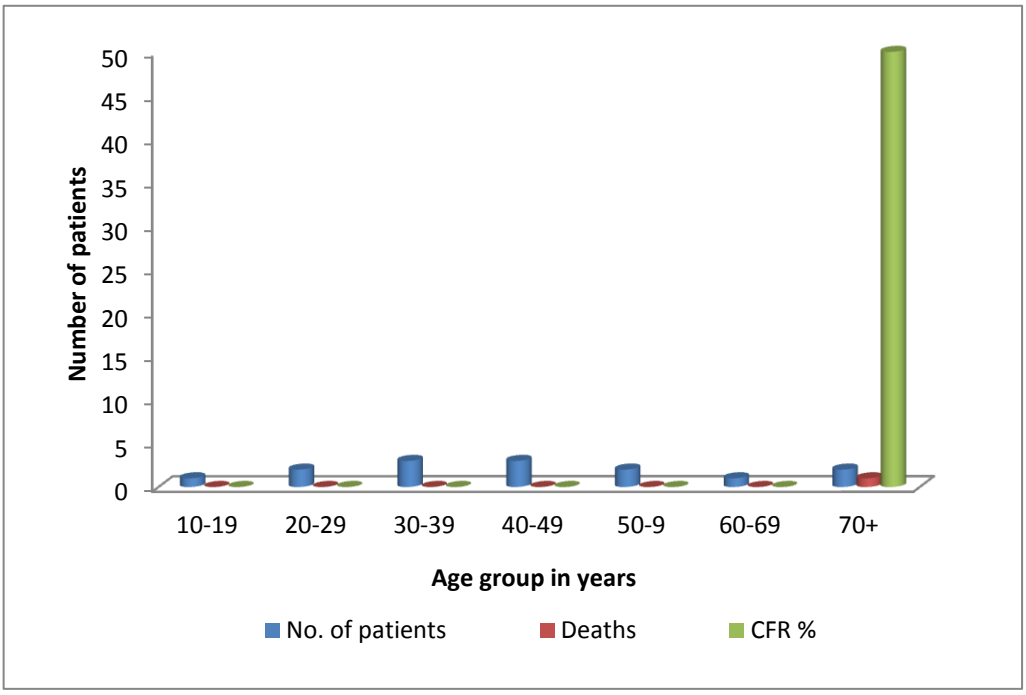


Fig III – CFR in females according to age and sex



SOURCE OF INFECTION AND CASE FATALITY RATE

The causes of intra abdominal sepsis were classified into 10 groups on the anatomical and functional basis as described by Meakins and associates.[3] The number of patients in each group and the respective mortality rate in each group are given in Table IV.

Table IV

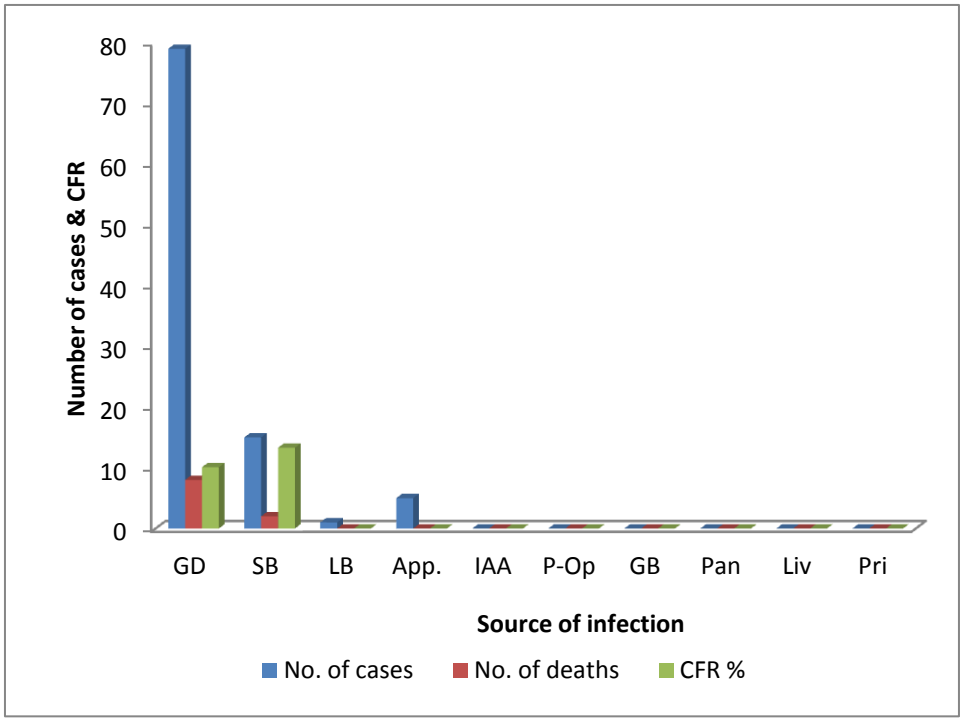
Source of Infection and CFR

Source	No. of cases	Percentage	No. of deaths	CFR %
Gastroduodenal	79	79	8	10.1
Small bowel	15	15	2	13.3
Large Bowel	1	1	0	0
Appendix	5	5	0	0
Intra abdominal abscesses	0	0	0	0
Post operative	0	0	0	0
Gall bladder	0	0	0	0
Pancreas	0	0	0	0
Liver	0	0	0	0
Primary peritonitis	0	0	0	0

All the patients had generalised peritonitis and abdominal infection was spontaneous in all of them. 79 patients had perforation of either gastric or duodenal ulcer, 15 patients had ileal perforation due to typhoid fever, 5 patients had appendicular perforation with generalised peritonitis and 1 patient had sigmoid volvulus with gangrene. There was no incidence of patients with peritonitis due to intra abdominal abscesses, gall bladder, pancreas and liver diseases, so also primary or post operative peritonitis in this study.

PHOTO :-A case of DU Perforation operated with septicaemia and wound dehiscence

Fig. IV – Source of infection and CFR



CD – Gastroduodenal

SB- Small bowel

LB - Large Bowel

App. - Appendicular

IAA - Intra abdominal abscesses

P-op - Post -operative

GB - Gall bladder

Pan. - Pancreas

Liv. - Liver

Pri. - Primary peritonitis

DURATION OF ILLNESS AND CASE FATALITY RATE

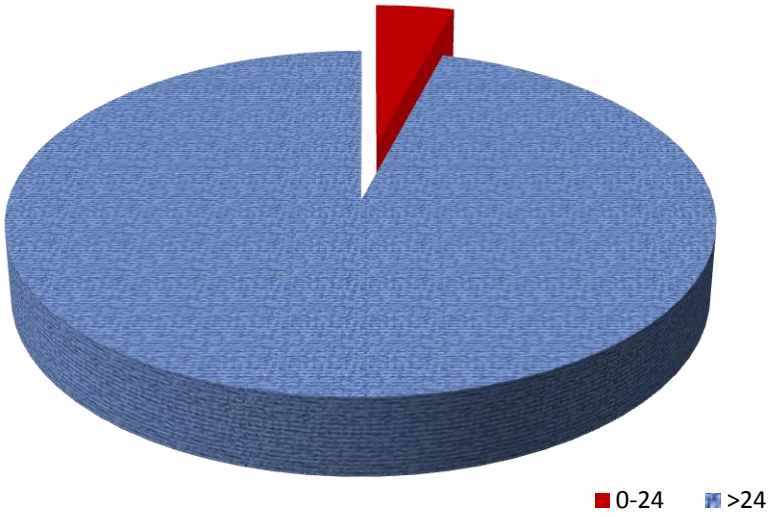
Table V shows the influence of duration of illness (from the onset of symptoms like pain abdomen to the commencement of treatment) on the outcome of intra abdominal sepsis. Analysis shows that those who underwent surgery after 24 hours had significantly raised CFR of 32.1% as compared to those who were treated before 24 hours (1.4%). The table also shows that there is a significant difference in CFR among different groups with a P value of < 0.05 which is significant.

Table V

Duration of illness and CFR

Duration of illness in hours	No. of patients	Deaths	CFR %
< 24	72	1	1.4
> 24	28	9	32.1

Fig. V – Duration of illness and CFR



APACHE II SCORE AND CASE FATALITY RATE

Table VI shows the relationship between APACHE II Score and CFR.

Table VI

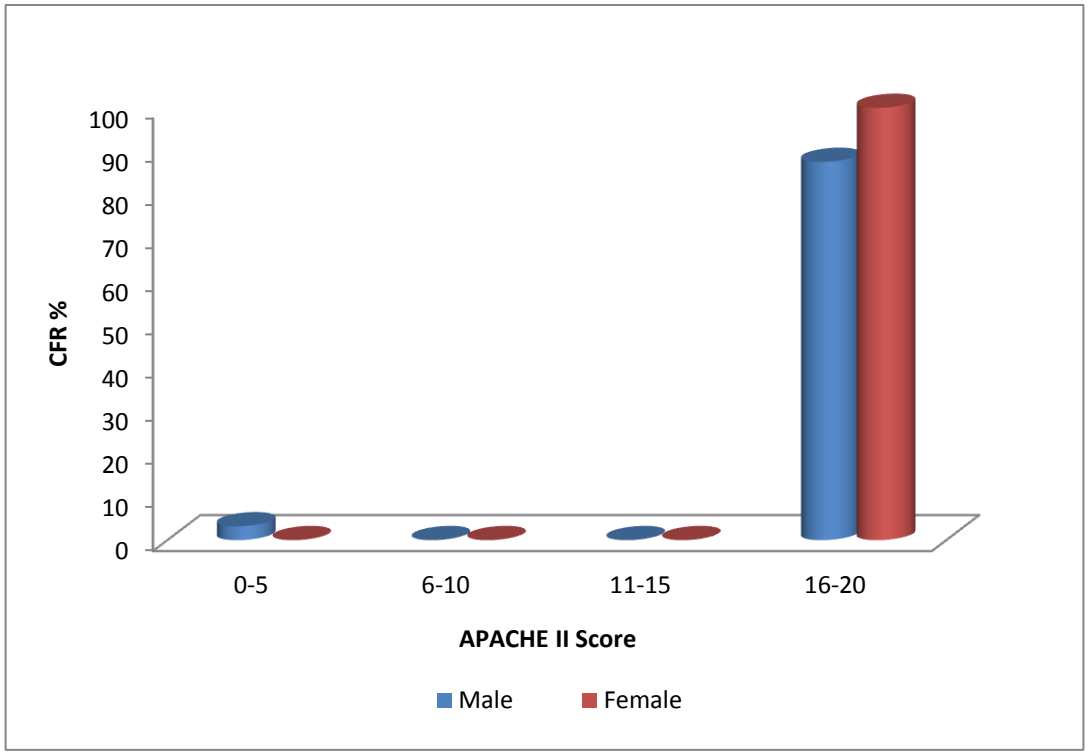
APACHE II Score and CFR

APACHE II Score	No. of patients		No. of deaths		CFR %		P
	Male	Female	Male	Female	Male	Female	
0-5	31	3	1	0	3.2	0	-
6-10	34	8	0	1	0	0	-
11-15	13	2	0	0	0	0	-
16-20	8	1	7	1	87.5	100	<0.05
Total	86	14	8	2	9.3	7	-

The overall mean APACHE II Score for 100 patients was 7.8 ranging from 1 to 20. The mean APACHE II Score in patients who expired was 14.8 compared to 7.1 in survivors. The maximum overall CFR of 88.9% was seen in patients with APACHE II Score of 16 and above. P value of less than 0.05 was observed here which was significant statistically.

An increase in APACHE II Score was associated with increased likelihood of mortality, as shown in the table below. There were 9 patients with APACHE II Score above 15 of which 8 expired with a CFR of 87.5% where as 91 patients had an APACHE II Score less than 15 of which 2 expired with a CFR of 2.2%. This is a significant prognostic factor in patients with intra abdominal sepsis.

Fig. VI - APACHE II Score and CFR



DISCUSSION

Peritonitis was recognized as uniformly fatal condition more than 2500 years ago. Surgical interventions was not attempted until early in the century but with the advancement in the medicine and intensive care, incidence of mortality was reduced by 50 percent.

Twenty years ago acute appendicitis appeared as the most common cause of peritonitis in most published figures. Next common causes of intra abdominal sepsis are perforated peptic ulcer (25%) post operative causes (10%) and gynecological causes (5)%). In many Indian studies typhoid perforation of the small bowel is also gaining importance. Most surgical centers are now finding that post operative causes are more worrisome.

Bohnen, Boulanger and their colleagues from Royal Victoria Hospital Montreal, in a study of 176 patients with intra abdominal sepsis reported a mortality rate of 38%.

Dellinger et al., 17 from Washington in a study of 187 patients with intra abdominal infection reported a mortality rate of 24%.

Kalfarentzos et al., 21 from Greece found that out of 42 cases, 19 died with an overall mortality of 45%

Pine et al.[11] reported that 32 of their 117 patients with intra abdominal sepsis died with mortality rate of 27%.

In the present study of 100 cases of intra abdominal seplsis due to various causes, 10 died. The overall mortality in this study was 10%.

Table VII

Overall CFR of Patients with Intra abdominal sepsis in different studies

Studies	Total No. of Patients	No. of Patients survived	No. of deaths	CFR %
Bohnen et al (1988)	100	69	31	31
Kalfarentzos et al (1987)	42	23	19	45.23
Dellinger et al (1985)	87	143	44	24
Bohnen et al (1983)	176	109	67	38
Pine et al (1980)	117	85	32	27
Present Study	100	90	10	10

AGE OF THE PATIENT AND CASE FATALITY RATE

The age of the patient also influences the mortality rate in intra abdominal sepsis. In the elderly, pre-existing conditions such as emphysema, diabetes or cardiovascular diseases compromise the ability to overcome the superimposed challenge of acute infection. The kidneys in elderly are more susceptible to the effects of stress.[43]

Bohen et al.,[20] in Itheir study of 176 patients found that patients above 50 years had a significant risk of death due to intra abdominal sepsis. In their study, patients less than 50 years old had a17% mortality whereas those over 50 years had a 45% mortality which was statistically significant.

Similarly Kalfarentzos et al.,[21] reported that in patients above 65 years, risk of death is significantly higher than in those below 65 years (P =< .05). In their study of 42 patients; 6 of 18 patients above 65 years died (33%).

Table VIII

Relationship between Age and CFR in various studies

	Bohnen et al (1988)	Kalfarentzos et al (1987)	Present Study
No. of Patients	176	42	100
Mean age of Patients expired	-	-	49.7
Cut off age	50 years	65 years	40
CFR above cut off age	45%	33%	16.3%
CFR below cut off age	17%	-	5.3%

In the present study the youngest patient to die of intra abdominal sepsis was a 13 year old male with ileal perforation secondary to enteric fever. The oldest patient to die was a 70 years old male with duodenal ulcer perforation. The mean age of those who died was 49.7 years in contrast to the mean age of those who survived which was 38.6 years. In this study patients were divided into two categories i.e those above 40 years and those below 40 years. In the group of 43 patients above 40 years, 7 died (16.3%) in contrast to 3 deaths out of 57 cases (5.3%) in the second group. This shows that there is a definite increase in the mortality of patients with intra abdominal sepsis above 40 years and this is statistically significant ($P = < 0.01$).

Various factors have been attributed to explain the increased risk of death in the elderly. They are:

1) Decreased Vascularity leading to decreased delivery of phagocytes.
2) Decrease in the number of mature T-Lymphocytes.
3) Decreased chemotactic and phagocytic activity of the polymorphonuclear leaukocytes.

DURATION OF ILLNESS AND CASE FATALITY RATE

Survival of patients with secondary peritonitis depends on many factors. Of paramount importance is the duration of peritoneal soiling before the leak is closed surgically or it seals off spontaneously.

Duration of the illness was measured in hours from the onset of illness to the time surgical treatment. It is however difficult to estimate the duration in post operative peritonitis.

Hunt[23] found that there was greater than two fold increase in morality in patients who underwent surgery after 24 hours of illness than in patients who under surgical intervention in less than 24 hours after the onset of symptoms. In their study of 44 patients with generalized peritonitis, only 17.6% of the patients who sought treatment in less than 24 after the onset of symptoms died compared to 50% of those patients who sought treatment more than 48 hours after the onset of illness.

Bohnen et al.,[20] also revealed that early surgical invervention within 24 hours will have better prognosis. In their study of 176 patients with generalized peritonitis, patients whose operative therapy was performed more than 24 hours after the onset of illness had a higher morality of 61& compared to 23% morality in those who underwent surgery within 24 hours. This difference in mortality was statistically significant ($p = < 0.005$).

TABLE IX

Relationship between duration of illness and CFR in various studies

	Hunt J.L. (1982)	Bohnen et al (1983)	Present Study
No. of Patients	44	176	100
Cut off time in hours from the onset of illness	48	24	24
CFR above cut off time	50%	61%	32.1%
CFR below cut off time	18%	23%	1.4%
P value		< 0.0005	< 0.05

In the present study all the 100 patients had spontaneous generalised peritonitis. Of them 93 underwent emergency laparotomy and 7 were brought in severe shock and were not fit for surgery. Of these 72 patients who underwent surgery within 24 hours from the onset of illness 1 patient died with CFR of 1.4%. Out of 28 patients who underwent surgery after 24 hours from the onset of illness, 9 died with CFR of 32.1%. Thus it can be seen that mortality rate rises significantly if surgery is delayed for more than 24 hours (P = < 0.01).

SOURCE OF INFECTION AND CASE FATALITY RATE

Although contamination can occur from a variety of causes, disruption of the gastrointestinal tract is the most common cause of intra abdominal sepsis.[7]

The source of contamination is an important prognostic factor. The reported mortality of peritonitis from a perforated duodenal ulcer or gastric ulcer ranges from 0-46%.[26] In contrast the mortality of peritonitis that originates from the small bowel usually by strangulation or obstruction, can be estimated at 20-25%.[26] Fortunately, the most frequent cause of peritonitis - perforated appendicitis - also has a rather low incidence of death.[26]

Perforation of the large bowel has a higher incidence of mortality.[26]

In the present study, the CFR in patients with peritonitis due to gastro- duodenal causes was 10.1% and increased to 13.3% as the cause proceeds to the small bowel.

Source of infection	Bohnen et al (1988)			Dellinger et ai (1985)			Present Study		
	No. of cases	No. of deaths	CFR %	No. of cases	No. of deaths	CFR %	No. of cases	No. of deaths	CFR %
Gastroduodenal	30	14	46	17	7	41	79	8	101
Small bowei	8	5	62.5	16	4	25	15	2	133
Large bowel	18	4	22	34	10	29	1	0	0
Post operative	4	1	25	80	23	29	0	0	0
Hepatobiliary	7	3	42	0	0	0	0	0	0
Pancreas	3	0	0	0	0	0	0	0	0
Appendix	15	0	0	40	0	0	5	0	0

APACHE II SCORE AND CASE FATALITY RATE

In this present study to estimate the risk of death of patients with intra abdominal sepsis APACHE II system was adopted. Knaus et al.,[38] stated that physiologic classification is more appropriate when assigned early in the patient's course independent of the effects of the treatment.

Several severity scoring are available. In the present study, the APACHE II score was selected because it consists of 12 liited values of routine physiologic measurements. Compared with the other scoring systems of Elebute and stoner and Stevens APACHE II is moreobjective and has been validated prospectively in large numbers of patients with a variety diagnosis. Other scoring are quite complex requiring considerable sophistication.

Bohnen et al.,[1] in their prospective study of 100 cases of abdominal sepsis report that an increase in APACHE II Score was associated with an increase likehood of mortality. Overall mean APACHE II score in the 100 patients was 13.72 with thw actual figures ranging from 0-36. The mean APACHE II in patients who died was 18.9, compared to 11.4 survivors.

Table XI

APACHE II Score and CFR

APACHE II Score	Bohnen et al (1988) (100 cases)			Present Study (100 cases)		
	No.of Patients	No.of death	CFR%	No.Of Patients	No.of deaths	CFR%
0-5	13	0	0	34	1	2.9
6-10	22	1	5	42	1	2.4
11-15	26	8	30	15	0	0
16-20	20	11	55	9	8	88.9
21-25	16	8	50	0	0	0
26-30	2	2	100	0	0	0
>30	1	1	100	0	0	0

In this present study the mean APACHE II Score was 7.8 with a range of 1 to 20 . The mean APSCHE II Score in patients who died was 14.8 compared to 7.1 in survivors Increase in APACHE II Score is directly proportional to the mortality rate. Above the score of 16 all patients died. The patients in the present study were divided into two groups i.e. with APACHE II Score upto 15 and with APACHE II Score greater than 15 had 88.9% morality compared to 2.2 % in Patients whose APACHE II Score was less than 15.

CONCLUSION

This prospective study was conducted in Tirunelveli Medical College Hospital, Tirunelveli during the 2011 to 2012. A total of 100 patients with proved intra abdominal who were admitted to general surgery wards during this period were included in this study. The mean age of the patients was 40.6 years and the overall mortality was 10 %.

The mean age of the patients who expired was 49.7 years. Patients above the cut of age of 40 years had a significantly higher mortality of 16.3% as compared to 5.3% among these below 40 years.

Duration of illness had a definite influence on the outcome of the disease. An early surgical intervention had a favorable effect with CFR as low as 1.4% in those who were operated within 24 hours of the onset of illness. Dalayed surgical intervention significantly raised the CFR to 32.1%

The cause of peritonitis also had its share of contribution in the outcome of the disease.CFR increased as the cause of peritonitis went down the gastro-intestinal tract starting with 10.1 % due to gastroduodenal cause and increasing to 13.3% due to causes in small beowel. There was no mortality in patients with appendicular pathology through the number of patients is very small to draw any conclusion.

No conclusion could be drawn regarding mortality in patients with large bowel pathology as there was only 1 patient in this group out of the 100 patients

studied. Similary nothing could be concluded regarding mortality due to post operative, pancreatic and primary causes as there were no patients in this study with such pathology.

The APACHE II Score was a very clear and significant indicator of the outcome of intra abdominal sepsis. Patients with an APACHE II Score more than 15 had 88.9% compared to 2.2% in those who had APAHCE II Score less than15. The higher CFR in patients with APACHE II Score more than 15 is constant with the results of other studies conducted previously.

No conclusion could be drawn regarding the effect of othe risk factors like DM, chronic respiratory and renal diseases on the outcome of patients with intra abdominal sepsis as none of the patients in this study had such risk factors.

In the end it can be stated that APACHE II Score , which is a reflection of the effect of various risk factors on the disease process in an individual, is a reliable indicator of the possible outcome in patients with intra abdominal sepsis. However the number of patients studied in this series as such is small compared to the high incidence of peritonitis and a larger number of patients need to be studied to come to a definite and statistically significant conclusion.

BIBLIOGRAPHY

1. Bohnen.J., Mustard R.A: Oxholm S.E:ET AT: 'APACHE II Score and Abdominal Sepsis : A Prospective study'. Arch Surg 123 : 225-229. 1988.

2. Bartlett J.G: 'The Paathophysiology of Intra-Abdominal Septis', In J.Mck.Watts; P.J.McDonald; P.E.O' Brien; V.R. Marshall &, J.J.Finlay jones (eds), Infection in Surgery: Basic and Clinical Aspects., London., Churchill Livingstone. 47-67, 1981.

3. Meakins J.L.Solomkin J.S.Allo M.D.et al: 'A Proposed classification of Intra abdominal infections'. Arch Surg 119 : 1372-1378, 1934.

4. Higgins G.M.et al as quoted by Hau T.Ahrenholz D.H.Simmons RL.1979.

5. Fowler 1900 as quoted by HauT., Ahrenholz D.H.Simmon R.L.1989.

6. Maddous M.A.Ahrenholz D.Z et al : 'The Biology of Peritonitis and implication for treatment'. Surg Clin North Am 68: 431-441, 1988.

7. Ahrenholz, D.H. : and Simmons, R.L., : Intra abdominal infection'. In Robert E.Condon and Jerome De Casse (Eds): Surgical Care II. Philadelphia, Lea & Febiger Page 283-295. 1985.

8. Altemeier W.A. ; Culbetson W.R.; Fuller, W;Shook, C; 'Intra abdominal Abcess'. Am J Surg : 125:70-79. 1973.

9. Rotstein O.D., Pruett T.L.Simmons R.L.; 'Lethal microbial synergism in intra abdominal infection'. Arch Surg 120: 146-151, 1985.

10. Clowes GHA Jr., Hirsch E.George BC., et al : Survival from sepsis: the significance of altered Protein metabolism regulated by Proteolysis including factor, the circulating cleavage Product of interleukin-1. Ann Surg 202 : 446-458. 1985.

11. Pine R.W.Wertz M.J., Lennard E.S., et a l; 'Determinants of organ malfunction or death in patients with Intra abdominal sepsis'. Arch Surg 118:242-249, 1983.

12. Bau A.E : Multiple, Progressive or sequential system failure : a syndrome of the 1970's. Arch Surg 110 : 779-781. 1975.

13. Fry D.E.Pearlstein L., Fulton R.L., et al : 'Multiple system organ failure the role of uncontrolled infection'. Arch Surg 115: 136-140, 1980.

14. Eiseman B.Beart R., Norton L: ' Multiple organ failure '. Surg Gynecol obstet. 144 : 323-326, 1977.

15. Fry D.E., : ' Multiple system organ failure'. Surg clin North 68 : 107-s122.1988.

16. Carico C.J.; Meakins J.L., Marchall J.C., et al : 'Multiple Organ Failure Syndrome'. Arch Surg 121 : 196-208. 1986.

17. Dellinger E.P., Werz M.J., Meakins J.L. et al: ' Surgical infection Stratification system for Intra-abdomjinal infection;. Arch Surg 120 : 21-29, 1985

18. Goris R.J.A., ; Theo P.A. te Boekhorst; Johannes K.S Nuytinck; et al : 'Multiple-organ Failure : Generalised Autodestructive inflammation ?'. Arch.Surg 120 :1109-1115, 1985.

19. Mohr J.A., ' Review of Pathophysiology' in Christian E.Kaufman Jr. & Soloman Papper (eds). Physical Consequences of Aging. 3-8, 1983.

20. Bohnen. J., ; Boulanger M., Meakins J.L., ; et al : 'Prognosis in Generalised Peritonitis : Relation to Cause and Risk factors'. Arch Surg 118: 285-290. 1983.

21. Kalfarentzos F.E Dougenis D.V., Cristopoulos D.C et al : ' Prognostic Criteria in Intra abdominal Sepsis'. Int.Surg 72: 185-187, 1987.

22. Fry D.E., Garrison R.N. Heitsh R.C., et al : ' Determinants of death in patients with intra abdominal abscesses'. Surgery 80: 517-523. 1980.

23. Hunt J.L., : ' General Peritonitis'. Arch Surg 17: 209-212, 1982.

24. Davis J.H., : ' Surgical Aspects of Diabetes Mellitus'. In David C.Sabiston Jr. (Ed), Text Book of Surgery. Editian 3, vol 1, Tokyo, W.B. Saunders Co., Page 151-158. 1986.

25. Hunt T.K.Ernest Jawetz: 'Inflammation, infection and antibiotics'. In Laerence W.Way (ed) Current Surgical Diagnosis and Treatment, Edition 8th, America, Printice Hall International Inc., 99-127, 1988.

26. Hau T., Ahrenholz D.H.Simmons R.L., : Secondary bacterial Peritonitis : The biologic basis of treatment'. Curr Probl Surg 16: 1-65, 1979.

27. Onodera T., Ouma M., Miura G., et al 1968: as quoted by Hau T., Ahrenholz D.H., Simmon R.L.1979.

28. Dawson J.L.1963 as quoted by Hau T., Ahreholz D.H.Simmon R.L., 1979.

29. Stephen M., Loewenthal J : ' Continuing peritoneal lavage in high risk peritonitis'. Surgery 85: 603-607, 1979.

30. Borzotta A.P., ; Polk H.C. Jr : ' Multiple system organ failure ' Surg Clin North Am.63 : 315-336 April 1983.

31. Machiedo G.W., Verma P.J., MC Govern P.J., et al : ' Patterns of Mortality in a Surgical intensive Care Unit'. Surg Gynecol obstet. 152: 757-795, 1981.

32. Sweet S.J.Glenmey C.U., Fitzgibbons J.P., et al: ' Synergistic effect of acute renal failure and respiratory failure in the Surgical intensive care unit' Am J Surg 141 : 492-496, 1981.

33. Stevens L.E., : ‘ Gauging the severity of Surgical Sepsis’. Arch Surg 118 : 1190-1192. 1983.

34. Stevens L.E., Clemme rT.B. Laub R.M.., et al : ‘ Fibronection in Severe sepsis’. Surg Gynecol Obstet. 162 : 222-228, 1986.

35. Skau T., Nystrom P.O., Carlsson C., : Severity of illness in intra abdominal infection : A Comparison of two indexes’. Arch Surg 120 : 152-158, 1985.

36. Elebute E.A., Stoner H.B., : ‘ The Grading of Sepsis” Br J Surg 70 : 29-31.1983.

37. Dominioni L.Dionigi R., Zenello M., et al : ‘Sepsis score and acute phase Protein repons as predictors of outcome in septic Surgical Patients’. Arch surg 122 : 141-146, 1987.

38. Knaus W.A., Zimmerman J.E., Wagner D.P., et al 1981 : as quoted by Dellinger E.P., 1988.

39. Andrus C., ; Doering M ; herrmann V.M., ; et al : ‘ Planned reoperation for generalized kintra abdominal infection ‘. Am J Surg 152 : 682-686.1986.

40. Altemeier, W.A., : ‘ Sepsis in Surgery’. Arch-Surg 117 : 107-112. 1982.

41. Dellinger E.P., : ‘ Use of Scoring systems to assess Patients with Surgical Sepsis’. Surg Clin N orth Am 68 : 123-145. 1988.

42. Jones P.F., : ‘ Acute Abdomen (1)’. In Selvin Taylor, Geoffrey D., Chisholm, et al (eds). Surgical Management. William Heinemann Medical book Ltd., London., 5, 1986.

43. Vincent J., Weil M., Puri V., : ‘ Circulatory Shock associated with Purluent Peritonitis’. Am J Surg 142 : 262-270, 1981.

44. Edwin A., Detch : ‘ Role of Intestinal Bones failure and Bacterial translocation’. In the development of Systemic infection and Multiple organ failure. Arch of S…… 1990 vol No. 125, Pgs, 403-404.

45. Rangabhashyam N. and Chandra Mohan S.M., Laparotomy in diffuse intra-abdominal sepsis – Recent advances in survey vol. IV pages 203-12.

46. Samptha Kumar P., Satyanaraya Rao C., ‘ Progbosis in intra-abdominal sepsis. Indian Journal of Gastroenteriology 1995, vol.14(1) Pgs.8-10.

47. Misra M.K., : ‘ The role of liver in multiple system organ failure’. Ind J Surg 51 : 451-467, 1989.

MASTER CHART

sl no	name	age	IP no	DOA	DOS	Rctal Tem	HR	MAP	RR	S Amylase	S Bicarbon	S Albumin	S Sodium	S Potassiu	S Creatini	HCT	WBC	C/S P FLD	APS	CHS	Age score	APACHE	Operative fin	procedure
1	PANJALIAMA	71/F	48733	20-10-2011	21-10-2011	37.5	64	123	26	41	28	5.6	130	4.2	1.2	32.3	14700	E.COLI	5	0	6	11	PER APND	LAP &AP DIS
2	SUBAIH	80/M	46671	21-08-2012	21-08-2012	37.6	100	123	20	247	12.7	2.9	128	4.9	1.6	36.9	2800	E & ST +	13	0	5	18	DU PER	PER CLO DEATH
3	SUDALAI	32/M	59512	20-12-2011	20-12-2011	38.7	80	125	24	190	24	4	128	4	1.1	46.9	6400	N.G	6	0	0	6	DU PER	PER CLO DIS
4	ADILAXMI	35/F	41774	13-01-2012	13-01-2012	37.5	104	100	22	175	13.6	4	126	4.5	1.5	39.2	1100	KLB	10	0	0	10	DU PER	PER CLO DIS
5	MARIAMMAL	45/F	22912	27-04-2012	27-04-2012	38.6	112	96	22	654	16.4	4	120	4.7	1.8	46.3	4900	E & STR	14	0	2	16	DU PER	PER CLO DEATH
6	POOMARI	30/F	56002	28-01-2011	28-01-2011	38.3	96	123.3	22	116	23	3	129	3.2	1.1	44.6	13400	N.G	5	0	0	5	DU PER	PER CLO DIS
7	SAMUAVEL	25/M	44975	26-09-2011	26-09-2011	37.2	86	143.3	20	136	26	3.8	121	3.6	1	39.5	17400	N.G	6	0	0	6	DU PER	PER CLO DIS
8	THANGARAJ	55/M	47720	27-08-2011	28-08-2011	372	140	146	24	660	13.6	3	135	3.8	1.6	45	1500	E.COLI	17	0	0	20	DU PER	PER CLO DIS
9	KATHANPILL	30/M	1930	11-01-2012	11-01-2012	37	80	126	22	114	25	2.6	129	4.6	1.2	39	17800	N.G	5	0	0	5	DU PER	B/L FLAN DIS
10	MARUDPANI	43/M	50449	01-11-2011	01-11-2011	37	82	124	22	299	28	3.4	135	4	1.4	42.8	12900	N.G	2	0	0	2	DU PER	PER CLO DIS
11	DASS	55/M	41378	07-09-2011	07-09-2011	37.2	80	120	22	166	23	3	124	4.2	1	50.7	11200	N.G	4	0	3	7	DU PER	PER CLO DIS
12	RAJA	35/M	3081	18-01-2012	18-01-2012	37	0	0	24	132	26	2.5	124	4.7	2.7	45.5	3000	N.G	11	0	0	11	DU PER	PER CLO DIS
13	ESAKIAMMA	22/F	3124	18-01-2012	18-01-2012	37.3	82	125	22	126	23	4	131	4.5	1.1	43	8900	N.G	2	0	0	2	PER APND	LAP &AP DIS
14	PALANI	30/M	2097	12-01-2012	12-01-2012	37.2	130	97	24	130	28	3.5	117	3.5	1.1	36	4800	KLB &E	5	0	0	5	JEJ PER	PER CLO DIS
15	SUDALAIMAI	32/M	48734	20-10-2011	20-10-2011	38	90	124	20	128	25	4.2	152	3.8	1	46.7	7800	N.G	4	0	0	4	DU PER	PER CLO DIS
16	ANANTH	45/M	27266	16-03-2012	17-03-2012	37	110	143	22	122	26	4	130	3.8	1	44.6	15900	E.COLI	6	0	2	8	ASC CLN	GRES &IT DIS
17	THANGARAJ	52/M	48216	11-10-2011	11-10-2011	37	78	90	22	62	26.5	4	137	3.9	1.3	38	2300	N.G	2	0	2	4	DU PER	PER CLO DIS
18	MUTHAIDHA	70/M	58390	14-12-2011	14-12-2011	38.3	88	84	22	312	19.2	3	130	4.8	1.3	48.4	14100	KLB &ST	4	0	5	9	DU PER	PER CLO DIS
19	PECTHIMUTH	55/M	58382	19-12-2011	22-12-2011	37.6	92	74	20	118	13.4	3.8	132	4.5	1.5	47.8	9900	N.G	3	0	3	6	GA PER	PER CLO DIS
20	SAMUAVEL	35/M	43452	21-08-2012	21-08-2012	37.4	86	78	21	152	20.8	3	127	3.6	1.2	43.7	6800	N.G	5	0	0	5	DU PER	PER CLO DIS
21	MAGESWAR	28/M	6234	03-02-2012	03-02-2012	37	84	80	22	142	28	3.2	128	4.5	1.6	48	6000	E.COLI	5	0	0	5	ILEAL PER	PER CLO DIS
22	ANNAMMAL	26/F	1781	10-01-2012	10-01-2012	38.1	88	76	20	221	28.2	3.6	125	4.3	1	37.5	10600	N.G	2	0	3	5	GA PER	PER CLO DIS
23	CHELPANDIA	50/M	60791	27-12-2011	27-12-2011	38.4	84	78	22	213	19.2	2.6	126	3.9	1.4	45.2	9100	E.COLI	5	0	2	7	DU PER	PER CLO DIS
24	ESWARAN	35/M	45640	14-08-2012	15-08-2012	38	78	86	22	123	19	3.9	135	4.6	1.6	52	13200	N.G	4	0	0	4	DU PER	PER CLO DIS
25	ARMUGAM	29/M	17556	30-03-2012	30-03-2012	38.1	92	78	29	262	18.6	3.8	139	3.7	1	50	2100	N.G	5	0	0	4	DU PER	PER CLO DIS
26	THANGARAJ	58/M	47720	27-08-2012	27-08-2012	38.2	94	88	23	169	23.2	3.3	136	4.6	1.2	48.7	9800	N.G	2	0	3	4	DU PER	PER CLO DIS
27	AYADURAI	45/M	50611	01-11-2011	05-11-2011	38	94	92	22	267	18.7	3.6	126	7.3	1.2	43	10900	PSEU	8	0	2	10	DU PER	PER CLO DIS
28	ARUMAGAM	30/M	41979	24-07-2012	25-07-2012	37.4	88	86	22	116	24	3.2	136	5.2	1.2	46.5	4700	E.COLI	2	0	0	2	DU PER	PER CLO DIS
29	RAMESH	14/M	47212	11-10-2011	11-10-2011	37.6	92	76	21	251	28.1	3	134	3.8	1.6	43.2	6200	N.G	3	0	0	3	DU PER	PER CLO DIS
30	INDRAN	35/M	42362	27-07-2012	28-07-2012	37	84	92	22	243	19	3.6	125	5.6	1.3	46	4800	E.COLI	6	0	0	6	DU PER	PER CLO DIS

sl no	name	age	IP no	DOA	DOS	Rctal Tem	HR	MAP	RR	S Amylase	S Bicarbor	S Albumin	S Sodium	S Potassiu	S Creatinii	HCT	WBC	C/S P FLD	APS	CHS	Age score	APACHE	Operative fi	procedure	results
31	ESSAKIMUTH	40/M	62661	27-12-2011	27-12-2011	38	92	84	26	122	19.5	3.6	122	5.6	3.1	47	4900	E.COLI	6	0	0	6	DU PER	PER CLO	DIS
32	MARIAPPAN	25/M	44250	06-08-2011	06-08-2011	37.2	78	82	20	116	22.5	2.8	131	4.5	1.2	46.5	3800	N.G	2	0	0	2	DU PER	PER CLO	DIS
33	RAJAN	59/M	46630	28-08-2012	28-08-2012	37.4	88	84	22	272	19.2	3.3	127	3.8	1.3	46	5400	PRT & COLI	4	0	3	7	DU PER	PER CLO	DIS
34	SUNDARAJAI	45/M	60067	23-12-2011	23-12-2011	37.8	88	92	22	241	26.4	3.2	133	5	1.1	46.8	7400	N.G	2	0	2	4	DU PER	PER CLO	DIS
35	ARUN	19/M	6099	06-02-2012	06-02-2012	36.7	96	113	24	112	22.8	3.7	128	3.8	1	52.8	30800	N.G	6	0	0	6	DU PER	PER CLO	DIS
36	MANIKANDA	35/M	18788	06-04-2012	06-04-2012	37.2	84	82	21	194	18.6	3.2	128	3.8	0.8	44.2	7800	PROTEUS	5	0	0	5	DU PER	PER CLO	DIS
37	CHINNAVEL	28/M	49290	24-10-2011	26-10-2011	37.8	76	88	21	252	20.2	3.7	132	4.5	1	45.2	18200	E.COLI	3	0	0	3	DU PER	PER CLO	DIS
38	BALMURGAN	38/M	28674	22-05-2012	22-05-2012	37.4	82	76	20	142	20.9	3.4	129	3.9	1.2	48.7	6700	N.G	6	0	0	6	DU PER	PER CLO	DIS
39	SUSAI	27/M	51952	03-12-2011	03-12-2011	38	72	73	20	148	25.4	2.8	122	4.6	1.6	48.1	6200	E.COLI	6	0	0	6	DU PER	PER CLO	DIS
40	NARAYAN	28/M	12581	23-03-2012	24-03-2012	37	98	74	26	220	21	3	126	3.8	1.6	47	2800	E.COLI	10	0	0	10	DU PER	PER CLO	DIS
41	PANDY	32/M	17469	30-03-2012	30-03-2012	37	102	103	24	157	26.2	3.6	123	5	1.2	39.9	9100	N.G	2	0	0	2	DU PER	PER CLO	DIS
42	PARAMSIVAN	45/M	14509	16-03-2012	16-03-2012	37.4	96	82	24	198	20.2	3	131	5.5	1	38.9	7700	E.COLI	4	0	2	6	DU PER	PER CLO	DIS
43	MANJUNATH	30/M	8823	15-02-2012	15-02-2012	37	84	78	18	112	18.8	3.6	125	4.4	1	46.5	8900	N.G	4	0	0	4	DU PER	PER CLO	DIS
44	INDUMATHI	22/F	3038	18-01-2012	20-01-2012	37.2	92	74	22	162	24.2	3.6	126	2.9	0.9	20.5	33900	PSEU & COL	8	0	0	8	DU PER	PER CLO	DIS
45	SUBRAMANI	45/M	9300	17-02-2012	17-02-2012	37.8	92	104	26	251	26	3.1	125	2.8	0.9	44.2	5400	E.COLI	6	0	2	8	DU PER	PER CLO	DIS
46	UDAYKUMAI	28/M	21136	18-04-2012	19-04-2012	37.4	100	93	22	452	18.4	3.1	118	4.7	2.1	46.8	15500	KLB &PROT	11	0	0	11	DU PER	PER CLO	DIS
47	RAMLINGAM	75/M	45916	16-08-2012	16-08-2012	38	90	0	28	167	22.5	4	135	4.6	0.8	46.3	10800	E.COLI	6	0	5	11	DU PER	PER CLO	DIS
48	RAJGOPAL	50/M	60759	29-12-2011	29-12-2011	37.2	100	96	30	192	21.5	3.9	125	3.5	2.5	48.4	3000	PSEU	9	0	2	11	DU PER	PER CLO	DIS
49	SUBAIH	55/M	41091	27-07-2012	28-07-2012	38.2	96	94	22	232	13.6	3.5	125	3.8	1.6	38.8	1500	KLB &PROT	10	0	3	13	DU PER	PER CLO	DIS
50	THIRUMALAI	30/M	18495	04-04-2012	05-04-2012	38.6	106	60	26	236	32.4	3.6	120	3.5	2	36.8	1500	N.G	12	0	0	12	ILEAL PER	PER CLO	DIS
51	ISSAC	35/M	30509	29-05-2012	30-05-2012	37.2	94	78	32	183	23.2	3.7	130	4.4	4.3	42.4	6500	N.G	5	0	0	5	DU PER	PER CLO	DIS
52	SELVARAJ	37/M	30387	29-05-2012	29-05-2012	38.1	84	78	20	212	26.2	3.1	127	3.4	1	50.1	2400	E.COLI	7	0	0	7	DU PER	PER CLO	DIS
53	KARTHIK	35/M	36611	19-06-2012	19-06-2012	38.6	102	72	25	332	18.4	3.1	128	5.3	1.2	44.3	10800	E & ST+	7	0	0	7	DU PER	PER CLO	DIS
54	YOKESH	35/M	35777	21-06-2012	21-06-2012	37.2	75	83	22	123	27.2	3.5	117	4	1	39.1	13000	N.G	3	0	0	3	DU PER	PER CLO	DIS
55	JESUDASS	35/M	35408	21-06-2012	22-06-2012	37.4	88	74	22	172	27.4	3.8	126	3.3	0.8	44.5	5900	N.G	3	0	0	3	DU PER	PER CLO	DIS
56	ANTONYMUT	35/M	48071	28-08-2012	28-08-2012	37	86	80	22	127	26.8	3	127	2.8	1.2	38.4	10900	N.G	3	0	0	3	DU PER	PER CLO	DIS
57	MD.ALI JINA	40/M	45611	14-08-2012	14-08-2012	37.2	110	0	22	142	26	3.2	129	4.7	3.2	50.7	1000	PSEU & COL	16	0	0	16	DU PER	B/L FLAN	DEAT
58	TANGPANDY	65/M	2711	16-01-2012	17-01-2012	38.5	100	84	23	351	19.4	3.1	132	4.6	1.3	64.2	8900	E.COLI	8	0	5	13	DU PER	PER CLO	DIS
59	TAMILRASI	30/F	39313	28-08-2011	28-08-2011	37	102	76	28	172	24	2.7	124	3.8	1.4	48.5	1500	E.COLI	9	0	0	9	ILEAL PER	PER CLO	DIS
60	KARPASAMY	40/M	16060	23-03-2012	23-03-2012	37.6	78	83	22	164	22.5	3	124	3.8	1.2	46.4	4200	N.G	4	0	0	4	DU PER	PER CLO	DIS

sl no	name	age	IP no	DOA	DOS	Rctal Tem	HR	MAP	RR	S Amylase	S Bicarbor	S Albumir	S Sodium	S Potassiu	S Creatinii	HCT	WBC	C/S P FLD	APS	CHS	Age score	APACHE	Operative fin	procedure	results
61	AMUDAN	46/M	45721	15-08-2012	15-08-2012	37.2	56	130	23	147	18.4	3.8	128	1.4	1.3	42.7	9600	N.G	9	0	2	11	DU PER	PER CLO	DIS
62	BALAJI	46/M	44393	07-08-2012	07-08-2012	38	0	0	28	163	20.2	3	136	4.2	1	66.5	4600	KLB &PRO	16	0	2	18	DU PER	B/L FLAN	DEATH
63	DASS	20/M	47068	23-08-2012	23-08-2012	38	92	73	24	106	33	2.9	135	2.8	1.3	47	2900	N.G	5	0	0	5	ILEAL PER	PER CLO	DIS
64	PANDY	13/M	38185	03-07-2012	04-07-2012	29.3	104	73.3	26	152	21.6	3	130	4.5	1.2	38.3	11000	N.G	4	0	0	4	ILEAL PER	RES & AI	DEATH
65	ASHOK	45/M	35824	21-06-2012	21-06-2012	37	8	78	22	201	36	2.8	138	3.8	1.6	32.5	6700	E.COLI	4	0	2	6	DU PER	PER CLO	DIS
66	MURUGAN	21/M	21148	18-04-2012	18-04-2012	37.2	80	96	24	997	19.2	4	122	4.1	1.2	43.8	15400	E.COLI	8	0	0	8	DU PER	PER CLO	DIS
67	LAXMANAN	40/M	32738	07-06-2012	07-06-2012	37.4	86	84	22	187	23.7	4	127	4.2	1.6	48.2	4100	N.G	5	0	0	5	DU PER	PER CLO	DIS
68	BRINDA	70/F	31833	04-06-2012	04-06-2012	37.4	80	83	24	43	23.4	4.2	125	4.8	1.7	35.8	15000	E.COLI	5	0	5	10	DU PER	PER CLO	DEATH
69	MUPIDATHY	28/M	43667	02-08-2012	02-08-2012	37.2	130	110	24	403	26.4	3.5	118	3.4	1.6	30.2	1700	E &STR	13	0	0	13	DU PER	PER CLO	DIS
70	INDIRA	35/F	23014	27-04-2012	28-04-2012	37.2	100	94	40	237	24.8	3.6	129	4.4	2	43.9	19800	STR &PRO	9	0	2	11	DU PER	PER CLO	DIS
71	ARUMAGSEL	65/F	53684	18-11-2011	18-11-2011	38.2	96	85	25	264	19.8	3.2	124	3.5	1.3	46.8	9400	N.G	7	0	3	10	DU PER	PER CLO	DIS
72	UDAYKUMAI	30/M	22978	04-05-2012	04-05-2012	37.2	104	63	22	123	24.2	3.8	126	5.8	1.7	46.9	4200	N.G	8	0	0	8	DU PER	PER CLO	DIS
73	CHELPERUM	50/M	21594	20-04-2012	20-04-2012	38.6	102	84	23	332	19.2	3.1	127	3.8	1.6	50.3	16300	E.COLI	11	0	2	13	DU PER	PER CLO	DIS
74	SURESHKUM	35/M	13341	24-02-2012	24-02-2012	37	0	0	28	367	22.3	3.2	114	4.9	1.6	47.4	2900	E.COLI	18	0	0	18	DU PER	B/L FLAN	DEATH
75	MURUGAN	65/M	22240	23-04-2012	23-04-2012	38.6	60	70	26	293	16.8	2.4	137	3.7	1.6	42.4	15700	E.COLI	12	0	5	17	ILEAL PER	PER CLO	DEATH
76	RAJMANI	45/M	21036	18-04-2012	18-04-2012	38.5	94	82	23	198	19.2	3.8	122	3.8	0.9	47.8	3200	N.G	5	0	2	7	DU PER	PER CLO	DIS
77	LAXMANSAN	33/M	18725	04-04-2012	04-04-2012	37.2	96	78	18	211	24.2	3.8	129	4.7	0.8	42.4	4600	N.G	2	0	0	2	GA PER	PER CLO	DIS
78	KOILPITCHAI	75/M	39079	03-07-2012	04-07-2012	38.4	98	86	22	231	23.1	2.9	122	4.5	2.1	39.9	15700	E.COLI	7	0	6	13	GA PER	PER CLO	DIS
79	KOILPITCHAI	65/M	37800	13-07-2012	16-07-2012	37.4	84	112	20	126	16.2	3.2	135	3.9	2.7	47.8	1800	E.COLI	12	0	5	17	DU PER	B/L FLAN	DEATH
80	THILAGAR	25/M	27584	17-05-2012	18-05-2012	37.4	84	88	20	116	34	3	138	4.5	1.3	48	7500	NO GROWTH	1	0	0	1	PER APND	LAP &AP	DIS
81	SANMUGAM	20/M	16714	26-03-2012	26-03-2012	38.4	115	73	24	325	22.9	3.2	136	5.5	1.6	43.8	2600	E.COLI	9	0	0	9	ILEAL PER	PER CLO	DIS
82	PONSAMY	42/M	5902	01-02-2012	01-02-2012	37.4	78	76	20	113	23.7	3.3	131	4.5	1.6	44.6	5800	N.G	3	0	0	3	DU PER	PER CLO	DIS
83	MARIMUTHU	35/M	8968	15-02-2012	15-02-2012	37.6	82	78	20	118	24.7	3.8	132	4.5	1.6	54.7	2800	KLB	6	0	0	6	DU PER	PER CLO	DIS
84	POOLIAMMA	59/F	35611	19-06-2012	19-06-2012	37.4	86	78	21	222	20.6	2.8	123	3.6	1.2	27.6	1800	E.COLI	7	0	3	10	DU PER	PER CLO	DIS
85	SUBAIH	60/M	6260	03-02-2012	03-02-2012	37.6	88	82	21	116	20.2	3.6	138	3.2	1	35	14200	KLB &STR	3	0	3	6	DU PER	PER CLO	DIS
86	MURUGAIH	52/M	40402	13-07-2012	13-07-2012	38.7	104	94	24	332	18.8	3	130	4	1.4	45.9	4200	N.G	4	0	2	6	ILEAL PER	PER CLO	DIS
87	GUNASELAN	18/M	8809	15-02-2012	16-02-2012	38.7	108	88	26	126	19.4	3.1	129	4.3	1.4	40.8	16300	E.COLI	8	0	0	8	ILEAL PER	RES &AN	DIS
88	EMANUAL	40/M	9897	21-02-2012	21-02-2012	37.2	108	90	22	126	30.1	3	129	4.6	1.8	42	16000	E.COLI	6	0	0	6	DU PER	PER CLO	DIS
89	SELVAM	20/M	3127	18-01-2012	18-01-2012	38	84	78	22	117	22.8	3.4	118	4.6	1.2	45.4	7100	PRT & COLI	4	0	0	4	ILEAL PER	PER CLO	DIS
90	KOILPITCHAI	60/M	18036	02-04-2012	02-04-2012	37	76	104	20	133	26.8	3.6	133	5.2	2.4	38.5	7700	N.G	3	0	3	6	DU PER	PER CLO	DIS
91	VENKATESH	55/M	58282	13-12-2011	15-12-2011	37.2	104	89	22	236	19.6	2.8	131	3.8	1.4	47.3	1900	E.COLI	6	0	3	9	DU PER	PER CLO	DIS
92	PETCHIAMAI	36/F	44960	26-09-2011	27-09-2011	37	110	74	26	157	28.7	3.8	133	4.5	3.1	31.3	7000	N.G	6	0	0	6	DU PER	PER CLO	DIS
93	ANADSELVI	45/F	39117	23-08-2011	24-08-2011	39	94	93	22	152	24.5	3.4	131	4.7	1.6	33.1	3900	N.G	6	0	2	8	DU PER	PER CLO	DIS
94	RAMCHANDF	48/M	60058	23-12-2011	23-12-2011	40	92	90	26	432	26.7	3.1	117	4.6	2.1	50.4	1500	E.COLI	15	0	2	17	DU PER	B/L FLAN	DEATH
95	MUTHUSAM	13/M	58457	14-12-2011	14-12-2011	37.2	122	66	26	302	18.9	2.8	127	4.7	1	42.3	2700	E.COLI	12	0	0	12	ILEAL PER	PER CLO	DIS
96	SENTHIL	35/M	56459	02-12-2011	02-12-2011	37	82	84	26	127	18.8	3	114	3.2	1	45.1	12300	E &ST+	8	0	0	8	GA PER	PER CLO	DIS
97	MANIKANDA	35/M	42480	12-09-2011	13-09-2011	37	96	83	28	161	26.4	3.6	128	3.8	2	47	4800	E.COLI	7	0	0	8	DU PER	PER CLO	DIS
98	MARIAPPAN	29/M	44250	06-08-2012	06-08-2012	37.2	122	70	30	221	23.1	3.7	115	6.3	1.6	53.4	5800	E.COLI	13	0	0	13	DU PER	PER CLO	DIS
99	ARUMUGAM	35/M	40254	29-08-2011	29-08-2011	37.1	84	78	20	121	23.4	3.8	122	3.8	1.2	47.4	10700	N.G	3	0	0	3	DU PER	PER CLO	DIS
##	RAJENDRAN	30/M	39672	25-08-2011	25-08-2011	37.4	90	84	22	116	20.2	3	120	4.1	1.9	30.4	2400	E.COLI	9	0	0	9	DU PER	PER CLO	DIS

ABBREVATIONS:-

APNDX- Appendix

GA- gatric

IT ANA- ileotransverse anastomosis

DIS – discharge

PSEU- pseudomonas

STR – streptococci

E &ST+ - E.coli &staphyllococus coagulase +

CFR – case fatality rate

JEJ-jejunum

RES – resection

RES & ANA- resection & anastomosis

N.G – no growth

PRO – proteas

ST- staphylococcus

KLB –klabsella

DATA SHEET

DATA SHEET / PROTOCOL

Name :

Age :

Sex :

I.P.No :

Name of the Hospital :

Address :

Date of Admission :

Date of Surgery :

Date of Discharge :

Signs & Symptoms :

Previous History of Surgery :

Co-Morbidity :

Medical Illness - C.V.S

- R.S
- Renal
- C.N.S

Examinations :

Vitals : Pulse rate

: B.P

: Resp. Rate

: Heart Rate

Temperature (Rectal) :

G.C.S

Investigations : C.B.C

: L.F.T

: Urine (A,S,D)

: Blood (Sugar, Urea, Creatine)

: Ser.Na^+ Ser.K^+

: Haemotocrit

: Ser.Amylase Ser. Lactate

: Ser.Alk.Phosphate Ser.Bicorbonate

: X-ray Abdomen

: USG Abdomen

: C/S Peritoneal Fluid

Treatment Received :

Acute Physiological Scoring

PARAMEITER	**VALUES**	**POINTS**
Rectal temperature		
Heart Rate		
Respiratory Rate		
Mean Arterial B.P		
Ser.Amylase		
Ser. Bicarbonate		
Ser. Albumin		
Ser. Sodium		
Ser. K+		

Ser. Creatinine		
Haematocrit		
W.B.C		

Acute Physiological Score (A)

Age Score (B)

Chronic Health Score (C)

Total Scoring = A+B+C